

Institute of Plant Nutrition and Soil Science

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Influence of the sulphur and nitrogen supply on S metabolites involved in Sulphur Induced Resistance (SIR) of *Brassica napus* L.

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In the memory of my father

TABLE OF CONTENTS

<i>Table of contents</i>	<i>I</i>
<i>List of figures</i>	<i>III</i>
<i>List of tables</i>	<i>VI</i>
<i>List of tables in the appendix</i>	<i>VIII</i>
<i>Abbreviations</i>	<i>IX</i>
1 Introduction	1
1.1 Sulphur metabolism of higher plants.....	3
1.2 The concept of Sulphur Induced Resistance (<i>SIR</i>).....	6
1.3 Objective of the work	11
2 Material and methods	13
2.1 Experimental sites.....	13
2.2 Experimental design	18
2.3 Sampling procedures	21
2.4 Plant analysis	21
2.4.1 <i>Mineral nutrients</i>	22
2.4.2 <i>Organic sulphur compounds</i>	23
2.5 Disease assessment.....	25
2.6 Statistical analysis.....	26
2.6.1 <i>Standard statistical analysis</i>	26
2.6.2 <i>Geostatistical analysis</i>	27
3 Results	31
3.1 Influence of sulphur and nitrogen fertilisation, cultivar and fungicide treatment on mineral composition and yield of winter oilseed rape.....	31
3.2 Influence of sulphur and nitrogen fertilisation, cultivar and fungicide treatment on sulphur-containing primary and secondary compounds of winter oilseed rape	42
3.3 Influence of sulphur applications on the level of fungal infections in winter oilseed rape crop	58

3.4	Changes in metabolite concentrations and enzymatic activities in response to <i>Pyrenopeziza brassicae</i> infection.....	67
3.5	Spatial patterns for total sulphur and sulphur-containing compounds in dependence on infections caused by <i>Leptosphaeria maculans</i>	76
4	<i>Discussion</i>	83
4.1	Relationship between S supply, productivity and mineral nutrient status	84
4.2	Significance of cultivar, sulphur and nitrogen fertilisation on sulphur-containing primary and secondary compounds of winter oilseed rape with view to <i>SIR</i>	91
4.3	Significance of the sulphur nutrition for crop resistance against fungal pathogens.....	97
4.4	Influence of infections by <i>Pyrenopeziza brassicae</i> on metabolite concentrations and enzymatic activities	100
4.5	Application of geostatistical analysis for the identification and interpretation of infections caused by <i>Leptosphaeria maculans</i>	107
4.6	Verification of the <i>SIR</i> concept employing field experimental data from different sites	110
5	<i>Summary</i>	115
6	<i>References</i>	121
7	<i>Appendix</i>	<i>i</i>

1 Introduction

Agricultural systems world-wide face several critical challenges: in developing countries the major need is to increase productivity in order to provide food for a growing population, while in industrial countries the quality of foodstuff next to environmental aspects of agricultural production is of prime interest. Plant nutrition is essential for maintaining crop productivity and yielding high quality feedstuff. Beside nitrogen (N), sulphur (S) plays a vital role in agriculture as an essential nutrient in the regulation of plant growth and development. Up to 90% of the total S in plants is bound in the amino acids cysteine, cystine and methionine (Giovanelli et al., 1980). In addition, S is a component of physiological important compounds such as glutathione, co-enzymes (biotin, thiamine pyrophosphate, CoASH), iron-S proteins (ferredoxins, thioredoxins), sulpholipids, glucosinolates, and polysaccharides (Thompson et al., 1986).

Until the 1980's S was regarded as the yellow poison and rapidly associated with the "acid rain" phenomenon (Schnug, 1998). The significance of S as a plant nutrient received only little attention compared to other macronutrients because the supply from the atmosphere (S depositions) and fertilisers was sufficient to meet crop requirements. With increasing utilisation of fossil fuels along with industrial and urban development, S emissions into the atmosphere increased steadily and reached its maximum in the 1970's with values of about 45-50 kg S ha⁻¹ in Germany (Schnug et al., 1993a). Because of the damaging effect of high SO₂ concentrations on plants and human health, various international protocols and agreements have come up to decrease S emissions (Schnug, 1998). The political consequences resulted in clean air acts in European countries and desulphurisation of industrial emissions. Changes in fertiliser practices towards S-free products and last but not least the break down of major emission capacities in the former east finally led to a drastic decrease of atmospheric S depositions in the beginning of the 1990's (Schnug et al., 1993a; Schnug and Haneklaus, 1994). In northern Germany, for instance, S depositions declined within only 12 years from about 40 kg S ha⁻¹ yr⁻¹ to 14 kg S ha⁻¹ yr⁻¹ in 1990 (Eriksen et al., 1998). As a result, the S supply of plants was not longer covered by atmospheric S depositions. Thus, symptoms of severe S deficiency became the most widespread nutrient disorder in field grown crops in northern areas of Europe (Schnug and Pissarek, 1982; Schnug, 1988). At the beginning of the 1980's, macroscopic symptoms of S deficiency were first reported for oilseed rape (Schnug and Pissarek, 1982) because of its high S demand (total S uptake: 41-94 kg S ha⁻¹; Haneklaus and Bloem, 2000). About ten-fifteen years later, symptoms of S deficiency were noticed in cereals and sugar beet, which have a significantly lower S demand with a total of 15-40 kg S ha⁻¹ and 10-20 kg S ha⁻¹, respectively (Schnug et al., 1993b; Schnug et al., 2000; Haneklaus and Bloem, 2000). Nowadays, severe S deficiency can be

observed regularly on production fields in northern Germany (Figure 1.1). Severe S deficiency has several consequences for the cultivation of arable crops: yield and quality are adversely affected (Schnug et al., 1993b; Schnug, 1997; Schnug and Haneklaus, 1998). Beside this, a decrease in the natural resistance of crops against some diseases has been observed in the northern part of Europe (Schnug and Ceynowa, 1990; Paul and Rawlinson, 1992; Schnug et al., 1995a) and in the UK (Lacey et al., 1987). For instance, in Scotland, where atmospheric S depositions are very low (Anon, 1990a; Dore et al., 2003), a natural infection of double-low oilseed rape cultivars by *Pyrenopeziza brassicae* (anamorph: *Cylindrosporium concentricum*; common name: light leaf spot) has been observed since 1984 when these varieties were first grown (Brokenshire et al., 1984). With decreasing S inputs to agro-ecosystems this disease has extended rapidly to the continent where it is known under the term “the Scottish disease” (Schnug and Ceynowa, 1990). Field trials carried out in Scotland, where a disease resistant and a non-resistant oilseed rape variety were treated with soil-applied S and foliar applied fungicide, showed that S fertilisation significantly improved plant health expressed in terms of crop yield, whereby this effect was comparable to fungicide applications (Schnug et al., 1995a). Thus, it was concluded that the S nutritional status interacts with the resistance mechanisms against this fungal disease (Schnug et al., 1995a). Induced resistance against crop pathogens by S fertilisation coined the term Sulphur Induced Resistance (*SIR*) (Schnug, 1997).



Figure 1.1: S deficient *Brassica napus* crop at main flowering (Immenrode, 2004; E 10° 43', N 51° 22') (photo: E Schnug).

The S metabolism of plants offers several options to tackle with biotic stress, including reduced gaseous S compounds (e.g. H₂S), cysteine, methionine, glutathione, glucosinolates, and phytoalexins (Schnug et al., 1995a; Haneklaus et al., 2002 and 2004). Earlier studies investigated the influence of the S supply on individual metabolites (De Kok et al., 1981; Schnug and Ceynowa, 1990; Schnug et al., 1995b; Haneklaus et al., 1999), while the interaction between metabolites in dependence on the S supply and in relation to the infection was only studied since this decade (Burandt et al., 2001; Bloem et al., 2004; Haneklaus et al., 2004; Salac et al., 2004).

Cysteine, glutathione and glucosinolates are supposed to be the best candidates for *SIR* because their concentration in plant parts and tissues proved to be closely related to the S supply (De Kok et al., 1981; Schnug et al., 1995b; Wielebski et al., 1999). The H₂S release on the leaf surface is also supposed to be an effective part of the plant defence system against fungal pathogens (Schnug et al., 1995a; Burandt et al., 2001). Therefore, in this study particular attention has been paid to these S-containing compounds.

1.1 Sulphur metabolism of higher plants

The assimilation of S in plants takes place in four consecutive steps: (1) the uptake of sulphate (SO₄²⁻) by the roots and transport of SO₄²⁻ throughout the stem (in the xylem vessels) to the leaves; (2) the activation of inorganic SO₄²⁻ by ATP and catalysed by ATP-sulphurylase or APS-kinase, enzymes that yield the sulphate-activated forms APS (adenosine-5'-phosphosulphate) and PAPS (3'-phosphoadenosine-5'-phosphosulphate), respectively; (3) the reduction of the activated SO₄²⁻ to sulphide (S²⁻); (4) the incorporation of S²⁻ into L-cysteine (Figure 1.2). There have been contradictory reports on S assimilation pathways in plants (Wray et al., 1998; Leustek and Saito, 1999), but recent investigations seem to resolve the old controversy about "free sulphite" and "bound sulphite" routes (Figure 1.2). Most of the results pointed out APS reductase (pathway 3) as the enzyme possessing most control over the flux through sulphate assimilation in plants (Suter et al., 2000). For a deeper treatise of the S metabolism several books and reviews are available, e.g. Brunold and Rennenberg (1997), Leustek and Saito (1999), Leustek et al. (2000), Hell (2002). For more details on the biochemical characterisation, localisation and genomic organisation of the enzymes involved in S assimilation see Kopriva and Koprivova (2003).

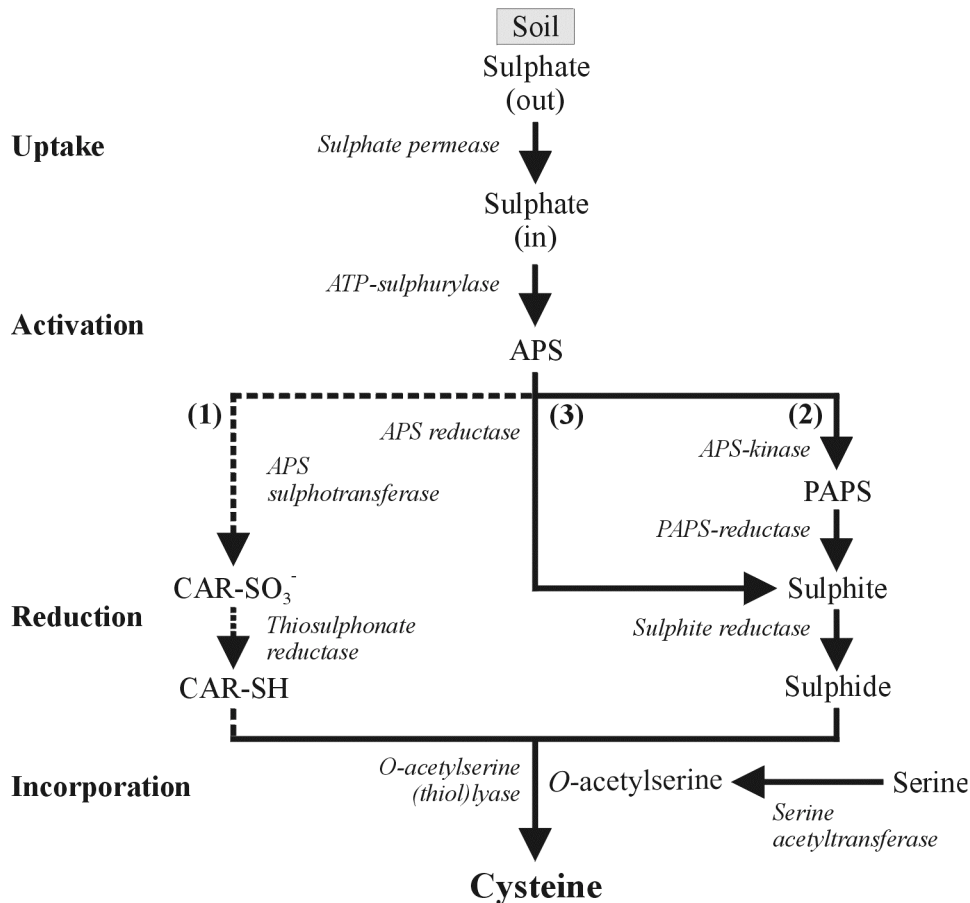


Figure 1.2: Sulphate assimilation pathways in higher plants: (1) APS reduction pathway or “bound” sulphite pathway; (2) PAPS reduction pathway or “free” sulphite pathway, and (3) direct reduction of APS to free sulphite, without prior activation to PAPS (according to Barroso et al., 1997; Kopriva and Koprivova, 2003).

Cysteine is the first stable organic product in which S is present in a reduced form. The biosynthesis of L-cysteine by plants is considered a key biological process because animals are not able to synthesise S-containing amino acids and thus, they have to take them up with the diet. The free cysteine pool was identified as one factor involved in plant resistance (Vidhyasekaran, 2000) and non-protein cysteine as a precursor of all relevant S-containing compounds putatively involved in *SIR* such as glutathione, glucosinolates and H_2S .

Cysteine synthesis

The incorporation of S^{2-} into cysteine proceeds through two enzymatic reactions (Figure 1.2). The first is catalysed by serine acetyltransferase (SAT), whereby L-serine is acetylated by acetyl-CoA to form *O*-acetylserine (*OAS*). *OAS* is considered to be the metabolic link between S and N assimilation pathways and possibly regulates cysteine biosynthesis (Giovannelli, 1990;

Leustek and Saito, 1999; Kopriva and Koprivova, 2003). The second reaction is catalysed by *O*-acetylserine(thiol)lyase (*OAS*-TL), in which *OAS* reacts with S^{2-} to form L-cysteine. SAT and *OAS*-TL form a multi-enzyme complex in bacteria (Kredich and Tomkins, 1966) and plants (Droux et al., 1998; Wirtz et al., 2001) known as cysteine synthase. In higher plants these enzymes were found in all three compartments involved in protein synthesis: cytosol, chloroplasts and mitochondria (Droux et al., 1998; Wirtz et al., 2001).

Hydrogen sulphide synthesis

The emission of hydrogen sulphide (H_2S) from higher plants has been reported in several studies (Filner et al., 1984; Rennenberg, 1989; Schroeder, 1993). The H_2S release may take place before or after cysteine synthesis (Rennenberg, 1983 and 1989). *OAS*-TL acts as a catalyst for the synthesis of cysteine from *OAS* and S^{2-} (see above), but may also release H_2S in a side reaction (Schmidt 1977a and 1977b). The degradation of cysteine by L-cysteine desulphhydrase (LCD) to H_2S and alanine or to H_2S , ammonium and pyruvate has been reported in numerous bacteria and higher plants (Rennenberg et al., 1987; Giovanelli et al., 1980; Burandt et al., 2001).

Glutathione synthesis

Glutathione (L- γ -glutamyl-L-cysteinyl-glycine) (GSH) is a S-containing compound of the primary metabolism and is the most abundant low molecular weight non-protein thiol in the plant. Glutathione is predominantly present in its reduced form and represents a form of storage and transport of reduced S in plants. At the same time, GSH controls sulphate influx into the plant at the level of sulphate uptake and xylem loading in the roots (Rennenberg and Herschbach, 1995; Barroso et al., 1997). Glutathione is synthesised in a two-steps enzymes-catalysed reaction at the extent of ATP. In the first step, γ -glutamyl-cysteine is produced from L-glutamate and L-cysteine in a reaction catalysed by γ -glutamyl-cysteine synthetase (γ -ECS). In the second step, catalysed by glutathione synthetase (GS), glycine is added to γ -glutamyl-cysteine at the C-terminal site to yield GSH. For a detailed description of the GSH synthesis see Brunold and Rennenberg (1997).

Glucosinolate synthesis

Glucosinolates (GSLs) are low molecular mass S and N-containing secondary metabolites that occur in at least 15 dicotyledonous taxa. Within this group, the *Brassicaceae* accounts for most species of agricultural relevance (Schnug, 1993). Glucosinolates are supposed to play a role as an intermediary metabolic S storage via an enzymatic recycling (Schnug, 1990 and 1993;

Schnug and Haneklaus, 1993a). After enzymatic cleavage, GSLs yield thiocyanates and sulphate, which can be further utilised for the synthesis of primary products (e.g. cysteine, methionine, GSH) under conditions of S starvation (Schnug, 1990 and 1993). Their basic structure is synthesised from α -amino acids, e.g. methionine in the case of alkenyl, thio, sulphinyl and sulphonyl glucosinolates; tryptophan in the case of indole glucosinolates. The first stable products in the GSL biosynthetic pathway are hydroxylated amino acids, which are the precursors of aldoximes (Underhill, 1980). In the next step, the thiol group of cysteine is transferred to aldoxime synthesising a thiohydroximic acid. The thiohydroximic acid is then glucosylated by the action of thiohydroximate-glucosyltransferase to lead to desulphoglucosinolates. After the transfer of sulphate from PAPS by a sulphotransferase, GSLs derive. From this basic structure different GSLs are derived by the action of specific enzymes through elongation and hydroxylation of the side chain. Further information on the GSL synthesis may be found in several books and reviews, e.g. Underhill (1980), Schnug (1990), Rosa et al. (1997), Selmar (1999) and Falk et al. (2004).

1.2 The concept of Sulphur Induced Resistance (SIR)

The fungicidal effect of elemental S on pests and diseases has been exploited in agricultural production since the end of the nineteenth century (Hoy, 1987). Foliar applied elemental S has proved to be most efficient against *Uncinula necator* (powdery mildew) (Coleno, 1987; Cook, 1987; Hoy, 1987; Bourbos et al., 2000; Reuveni, 2001; Kassemeyer, 2003), but was also successfully used against other pathogens such as *Erysiphe graminis* (downy mildew) in cereals (Hoy, 1987), *Streptomyces scabies* (common scab) in potato (Vlitos and Hooker, 1951; Mortvedt et al. 1963), and *Alternaria brassicae* (alternaria black spot) in oilseed rape (Anon, 1988). In comparison, the significance of soil-applied S for plant resistance, independent on the S form, only became evident a century later. In S deficient environments, soil-applied S fertilisation significantly reduced infections of oilseed rape by *P. brassicae* (Schnug et al., 1995a), of grapes by *U. necator* (Bourbos et al., 2000), and of potato by *Rhizoctonia solani* (stem canker) (Klikocka et al., 2004). Also in greenhouse experiments under controlled conditions the relationship between S nutrition and fungal infections could be demonstrated. For instance, Wang et al. (2003) showed that increasing rates of soil-applied S as ammonium sulphate decreased the disease index of young plants (4 to 6 leaves) by 5%, 21% and 44% for infections of oilseed rape by *Sclerotinia sclerotiorum* (sclerotinia stem rot), of corn by *Bipolaris maydis* (corn leaf blight) and of winter wheat by *Rhizoctonia cerealis* (sharp eyespot), respectively.

The results from greenhouse and field experimentations clearly demonstrated an interaction between S supply and plant diseases for different crop plants, whereby the underlying mechanisms remained obscure. The fungicidal effect of foliar applied elemental S has to be strictly distinguished from the health promoting effect of soil-applied S, but the mode of action shows marked parallels to different plant metabolites putatively connected with *SIR* (Haneklaus et al., 2004).

The S metabolism offers several efficient mechanisms by which plants are able to cope with fungal attacks. Investigations of Burandt et al. (2001) provided the first evidence that the evolution of H₂S, and therefore the S nutrition, plays an important role in the plant natural defence system against fungal infections. Other mechanisms to tackle with biotic stress that are provided by the S metabolism of plants include GSH, GSLs and phytoalexins (i.e. low-molecular weight antibiotics, pathogenesis-related proteins and elemental S formation) (Haneklaus et al., 2002 and 2004). The S-containing compounds, which are supposedly involved in *SIR*, are summarised in Table 1.1. Among these pathogen-directed defence compounds, enzymes involved in the H₂S release, GSH and GSLs have received attention as putative defences in the present study and are outlined in the next lines with respect to their possible role in *SIR*. Phytoalexins are also known as a primary factor in plant defences (Sinha, 1995), but their role in *SIR* still needs to be proved.

Taking into account that cysteine is the precursor of all relevant S-containing metabolites putatively involved in *SIR* (see chapter 1.1), it might be assumed that cysteine is one of the cornerstones of the plant resistance against pathogens. Vidhyasekaran (2000) reported that cysteine and methionine are enriched in resistant plant tissues. Thus, these amino acids may be related to the resistance of plants against pathogens. Cysteine occurs both in the free state and bound to proteins in plant tissues and it is strongly related to the S nutritional status of plants (Bosma et al., 1990; De Kok, 1990). Under conditions of S deficiency a decrease of S-containing amino acids in proteins was observed (Schnug, 1997). This implies that a sufficient S supply might be related to increased resistance of plants against pathogens through elevated cysteine levels, since cysteine may be itself involved in pathogenesis or the rate of cysteine synthesis may be tailored to the demand of other S-containing defence compounds.

Table 1.1: Influence of the S supply on the concentration of S-containing metabolic compounds and their possible roles in *SIR* (according to Haneklaus et al., 2002).

<i>S</i> -containing metabolites	Indications for interactions between <i>S</i> supply and <i>S</i> metabolites	Phytopathological effect
Reduced gaseous S compounds	Genotypical differences for H ₂ S releasing enzyme activities (Burandt et al., 2001); dependence of enzymatic activities on infection (Bloem et al., 2004)	fungitoxic
Glutathione (GSH)	Positive significant relationship in pot experiments (De Kok et al., 1981; Schnug et al., 1995b)	anti-oxidative effect; presumably messenger in the hypersensitive reaction (Foyer and Rennenberg, 2000)
Glucosinolates (GSLs)	Positive significant relationship in vegetative and generative plant parts (Schnug, 1989 and 1997)	qualitative defence mechanism (Rosenthal and Janzen, 1979); retarded multiplication of spores, but no impact on growth of the mycelium (Drobznica et al., 1967)
Low-molecular weight antibiotics	Speculative on basis of data for precursors	fungitoxic
Pathogenesis-related proteins	Speculative on basis of relationship for protein content and composition (Schnug, 1997)	toxic for micro-organisms and insects
Elemental S formation	Genotypical differences for tomato in dependence on resistance against <i>Verticillium dahliae</i> (Williams et al., 2000)	fungitoxic

Hydrogen sulphide releasing enzymes

H₂S is known as a fungitoxic gas, so that the H₂S emission from higher plants may be one effective defence mechanism involved in plant resistance (Table 1.1) (Schnug et al., 1995a). Beffa (1993) found a fungicidal effect of elemental S on *Phomopsis viticola* spores before germination and attributed the mode of action to the reduction of elemental S by proteic and non-proteic sulphhydryl groups. Based on this investigation, it can be speculated that a minimum

uptake of $10 \mu\text{M H}_2\text{S h}^{-1}$ by the pathogen would be necessary to yield a fungicidal effect (Haneklaus et al., 2004).

So far the mechanism by which H_2S is released, the extent of H_2S emissions under natural conditions, the relationship to the S supply and infections by fungal pathogens still need to be verified. It is still discussed controversially if the enzymes LCD and OAS-TL are involved in the H_2S emission and if there is a relation between the enzyme activities and the S nutritional status of crops (Schmidt, 1977a and 1977b; Giovanelli et al., 1980; Burandt et al., 2001). Investigations of Giovanelli et al. (1980) showed that the cysteine synthase activity was insensitive to changes in the S supply and the LCD activity was not coupled to the emission of H_2S . Studies of Burandt et al. (2001) revealed a relationship between the S status of the plant and the LCD and OAS-TL activity. An increasing total S content of the plant was associated with decreasing LCD and increasing OAS-TL activity. An inverse relationship between enzyme activities was determined ($r = -0.675$; $p < 0.05$).

The LCD and OAS-TL activity may have a potential as an indicator for the H_2S emission if correlations between their activity and the H_2S release can be proved. It can be speculated that a fungal attack yields to an increased activity of LCD and/or OAS-TL and thus the release of fungitoxic H_2S . In the present research work, the influence of the S nutrition and fungal infections on the LCD and OAS-TL activity was investigated.

Glutathione

Numerous physiological functions have been attributed to GSH in the cellular plant metabolism (Rennenberg, 1995; Foyer and Rennenberg, 2000; Tausz et al., 2003). Beside its role in abiotic stresses (drought, low and high temperature, light, UV, salinity, anoxia, air pollution), GSH was also found to have an impact on plant/pathogen interactions (Table 1.1) (Gullner and Kömives, 2001). Studies of Vanacker et al. (2000) revealed that GSH accumulated rapidly in response to fungal attack, and this was proved to be decisive in pathogenesis (Gullner and Kömives, 2001). Increased GSH levels in cells surrounding the site of attack might have different roles in defence. Glutathione might increase protection from excessive damage caused by the accumulation of reactive oxygen species (ROS) during the oxidative burst (May et al., 1996). Changes in the redox state and concentration of GSH, as observed during the hypersensitive response (HR), might be an essential secondary messenger mediating the signalling effects of hydrogen peroxide (May et al., 1998; Vanacker et al., 1998; Foyer and Rennenberg, 2000). Hydrogen peroxide-mediated induction of GSH has been demonstrated in different systems such as *Arabidopsis* suspension cultures (May and Leaver, 1993) and tomato

plants (May et al., 1996). It is also supposed that the systemic induction of GSH-related antioxidative systems contributes to the development of systemic acquired resistance (SAR) against a subsequent infection (Fodor et al., 1997). Another option is the instant and reversible thiolation of proteins, which protects essential thiol groups in key proteins from irreversible inactivation during oxidative stress (Thomas et al., 1995).

Experiments have shown that S deficient plants have very low GSH concentrations and S fertilisation strongly increased the free thiol content (De Kok et al., 1981; Schnug et al., 1995b). Basically, S deficient plants are expected to be more vulnerable to stress factors, which are usually compensated by the GSH system, so that S fertilisation should have a positive effect on resistance mechanisms provided by the GSH pathways.

Glucosinolates

It is generally assumed that GSLs might be an important part of the constitutive defence mechanisms of GSL-containing plants against fungal pathogens (Table 1.1) (Rosenthal and Janzen, 1979; Greenhalgh and Mitchell, 1976; Mithen et al., 1986; Schnug and Ceynowa, 1990). Following cellular disruption, GSLs undergo hydrolysis catalysed by the enzyme myrosinase to produce an array of products of which isothiocyanates ('mustard oils') are a major component (Luethy and Matile, 1984). These compounds, and other products of GSL hydrolysis, have been shown to be toxic or inhibitory to many species of fungi and bacteria, including several *Brassica* pathogens such as *Alternaria* spp. (Doughty et al., 1991), *Leptosphaeria maculans* (anamorph: *Phoma lingam*; common name: stem canker) (Mithen et al., 1986 and 1987; Koch, 1989), and *Peronospora parasitica* (downy mildew) (Greenhalgh and Mitchell, 1976). *In vivo* studies revealed that GSLs accumulate in tissues as a result of infection by pathogens (Mithen et al., 1987; Koritsas et al., 1989; Birch et al., 1990; Schnug and Ceynowa, 1990; Doughty et al., 1991). The accumulation of GSLs in the infected parts of the plant might restrict the spread of existing fungal infections or inhibit subsequent attempted infections. However, many phytopathogenic fungi are still able to infect GSL-containing plants. It has been therefore speculated that fungal pathogens of *Brassica* have developed certain mechanisms to overcome GSL/myrosinase defence system by degradation or transformation of intact GSLs to non-toxic or less toxic products to prevent the release of toxic GSL degradation products catalysed by myrosinase (Giamoustaris and Mithen, 1997; Wu and Meijer, 1999; Sexton and Howlett, 2000). In this case, plants might develop other strategies for survival through remobilisation of sulphate. Thus, sulphate released by the fungal action can be utilised either for the synthesis of primary

products (see chapter 1.1) or can be released as H₂S, contributing in this way to resistance (Haneklaus et al., 2004).

The total GSL concentration in seeds as well as in vegetative parts of oilseed rape is closely related to the S nutritional status of the plant (Schnug, 1988; Schnug, 1989; Schnug et al., 1995b). Plants suffering from severe S deficiency might therefore be more susceptible to fungal diseases due to lowered GSL levels.

1.3 Objective of the work

Research on S induced resistance mechanisms against pathogens is limited and there are still open questions regarding the efficiency of S-containing metabolites in the activation and strengthening of plant defence responses, particularly under field conditions. A profound knowledge about the role of S-containing compounds in crop resistance to fungal pathogens and their triggers is vital for taking advantage of *SIR* by targeted fertiliser strategies. Then, the input of pesticides could be reduced or renounced. *SIR* may therefore become an important strategy to efficiently combat pathogens in conventional and organic farming. An important advantage of *SIR* compared to chemical pesticides is that the resistance will not be rapidly broken by new pathotypes.

The German Plant S Group (DFG-Forschergruppe 383), financed by the German Research Foundation (DFG), uses an integrated approach based on tools of molecular physiology and cell biology for the identification of the mechanisms causing *SIR*. The group in which the present research work was carried out is a member of the German Plant S Group (Teilprojekt 8 – Ewald Schnug, FAL, Braunschweig), investigating the agronomical aspects of *SIR*. Bilateral collaboration between the Institute of Plant Nutrition and Soil Science, FAL, Braunschweig and the Scottish Agricultural College (SAC), Aberdeen, Scotland had made possible parallel establishment of three field trials under different S nutritional conditions and different disease pressures, one in Braunschweig and two in Scotland.

In the present study, experiments were conducted in order to identify and evaluate the significance of S-containing metabolites for plant resistance against fungal pathogens under natural conditions. For a practical exploitation of *SIR* it is necessary to induce resistance consistently under field conditions because only then S fertiliser strategies, which prompt *SIR*, can be successful. Oilseed rape (*Brassica napus* L.), and here the varieties *Bristol* and *Lipton*, was chosen for experimentation. *Bristol* is the same cultivar used in the experiments of Schnug et al. (1995a) where *SIR* was reported for the first time. *Bristol* is susceptible to *P. brassicae*,

while *Lipton* is counted among varieties rated as resistant against this fungus. So that best conditions for identifying S metabolites involved in stress resistance were provided.

Pyrenopeziza brassicae is one of the most important diseases of oilseed rape crop in the UK (Lacey et al., 1987). Each year approximately £10 million are spent on fungicides to control fungal diseases, but despite this losses in excess of £48 million are attributed to *P. brassicae* (Fitt et al., 1997). Because of the devastating effect of *P. brassicae* on oilseed rape crop, in the present study particular attention has been given to this fungus.

The main objectives of the present research work were:

- I. To quantify the influence of S and N fertilisation, cultivar and fungicide treatment on mineral composition and S-containing primary and secondary compounds in winter oilseed rape (*Brassica napus* L.).
- II. To evaluate the significance of the S supply for infections of winter oilseed rape (*Brassica napus* L.) by fungal pathogens, with special view to *Pyrenopeziza brassicae*, *Leptosphaeria maculans* and *Peronospora parasitica*.
- III. To assess relationships between S-containing metabolites involved in *SIR* and fungal infections in dependence on the S supply.
- IV. To test different statistical methods in order to make relationships between S-containing metabolites involved in *SIR* and fungal infections obvious.
- V. To discuss the results of these field experimentations in the context of reported results along with a discussion on the importance of the present findings for *SIR* and possible practical applications of *SIR* in agriculture.

2 Material and methods

2.1 Experimental sites

Field experiments were conducted on sites with different atmospheric S depositions in Braunschweig (Germany), Aberdeen and Inverness (Scotland) in 2000-2003 (Table 2.1). In Scotland, field trials were carried out by the Scottish Agricultural College (SAC) in Aberdeen. Leaf and seed samples from these experiments were analysed in Braunschweig for mineral nutrients and S-containing primary and secondary compounds.

Table 2.1: General characteristics of the selected study sites.

Site	Location	GPS position	Soil type*	S deposition
Braunschweig	Germany	52° 18` N 10° 27` E	Cambisol	0.8-1.3 g S m ⁻² yr ⁻¹ (Beilke and Uhse, 1998)
Aberdeen	Scotland	57° 12` N 2° 13` W	Podzol	0.3-0.6 g S m ⁻² yr ⁻¹ (Dore et al., 2003)
Inverness	Scotland	57° 53` N 4° 5` W	Cambisol	< 0.3 g S m ⁻² yr ⁻¹ (Dore et al., 2003)

note: *FAO soil classification

Braunschweig

Braunschweig (E 10° 27', N 52° 18') is located in the northeast of Germany. The experimental field belongs to the experimental site of the Institute of Plant Nutrition and Soil Science of the Federal Agricultural Research Centre (FAL).

Braunschweig has a typical temperate climate, which is characterised by frequent changes in temperature, humidity, and winds. Prolonged warm spells in summer are infrequent. Meteorological records during the experimentation period were provided by the Braunschweig Experimental Station and are listed in the appendix (Table A.1). During experimentations, the mean temperature was about 9.2°C and the mean sum of sun hours about 1400 h. Precipitation records were 500 mm, 903 mm and 546 mm for the first, second and third season, respectively (Figure 2.1). The long-term mean annual precipitation for Braunschweig is 620 mm. Precipitation over winter is a major criterion for the occurrence of severe S deficiency and infections of winter oilseed rape by pathogens. In Figure 2.1, the precipitation rates during the growth period (September-July) and over fall-winter (September-February) are shown.

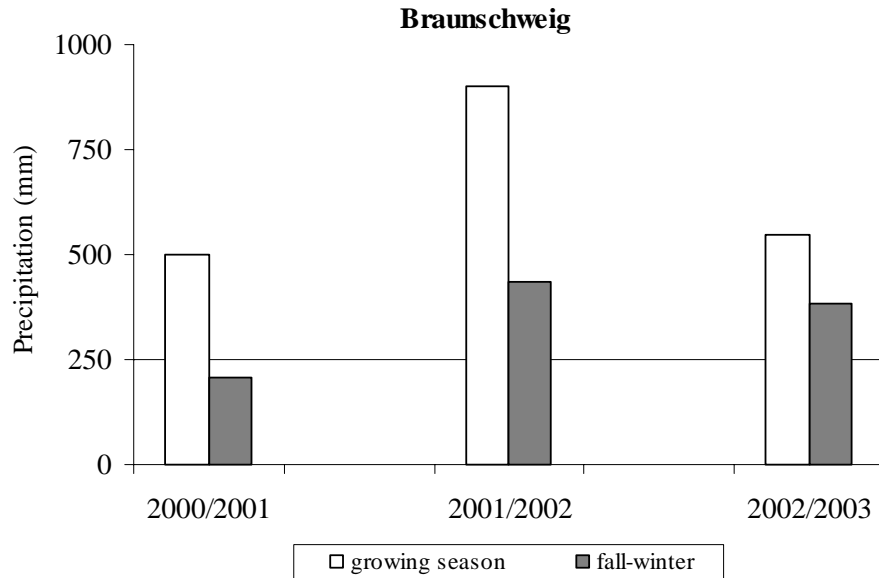


Figure 2.1: Precipitation (mm) during the growing season of winter oilseed rape and over fall-winter in Braunschweig.

Table 2.2: Description of soil parameters in Braunschweig (according to Rogasik et al., 2004).

Soil parameters	Method	Reference	Unit	Top soil	Sub soil
				(0-30 cm)	(30-60 cm)
pH	CaCl ₂	Hoffmann, 1991	-	5.5	4.8
Organic matter	dry combustion ¹	LECO Handbook	%	1.4	0.7
N_t	Kjeldahl	Hoffmann, 1991	%	0.08	0.05
P	CAL	Schüller, 1969	mg kg ⁻¹	61	47
SO₄-S	KCl	Bloem, 1998	mg kg ⁻¹	4.3	n.a. ²
K	CAL	Schüller, 1969	mg kg ⁻¹	94	90
Mg	CaCl ₂	Hoffmann, 1991	mg kg ⁻¹	39	38
Clay	areometer	De Leenheer et al., 1954	%	6.3	5.4
Silt	areometer	De Leenheer et al., 1954	%	46.7	47.2
Sand	areometer	De Leenheer et al., 1954	%	47	47.5

note: ¹analysis of C_t; organic matter = C_t*1.724; ²n.a. – not available

The physical and chemical characteristics of the soil at the experimental site in Braunschweig are summarised in Table 2.2. The soil type is a Cambisol with a loamy sand soil texture (< 6.5% clay; > 47% sand), characterised by low water retention capacity and high rates of leaching. The pH was found to range from highly acid (4.8) to moderately acid (5.5). The S status of the soil was low (Table 2.2).

Aberdeen

Aberdeen (W 2° 13', N 57° 12'), Scotland's third largest city, is located between Dee and Don rivers in the east part of Scotland. The Scottish climate is influenced by the convergence of warm subtropical and cold polar air masses, which generates a zone of cyclonic low pressure and an abundance of rain-bearing depressions. To a large extent the surface temperature of the North Sea influences the temperature in Scotland. The experimental site has a maritime climate. Meteorological data during the experimentation period were available from the local meteorological site at Dyce and are listed in the appendix (Table A.2). During experimentations, the mean temperature was about 8.3°C, the mean sum of sun hours about 1320 h and the rainfall ranged from 760 mm to 970 mm (Figure 2.2). The long-term mean annual precipitation for Aberdeen is 754 mm. Comparing precipitation rates recorded during the growth period (September-July) (882 mm, 763 mm and 825 mm for the first, second and third season, respectively; Figure 2.2) with the long-term mean annual precipitation, it can be stated that in all years the values were distinctly higher. As a result of such high precipitation rates during the growing season and particularly over fall-winter (September-February) (630 mm, 440 mm and 635 mm for the first, second and third season, respectively; Figure 2.2) and the generally light texture of podzols, it can be assumed that sulphate leaching on this site is larger than in Braunschweig.

At the Aberdeen experimental site the soil type is a humic Podzol with a mean soil pH of 6.2. Chemical soil characteristics are listed in Table 2.3. The soil can be described as a loamy sand soil characterised by a high organic matter content and sufficient levels of phosphorus (P), sulphur (S), potassium (K) and magnesium (Mg) (Table 2.3).

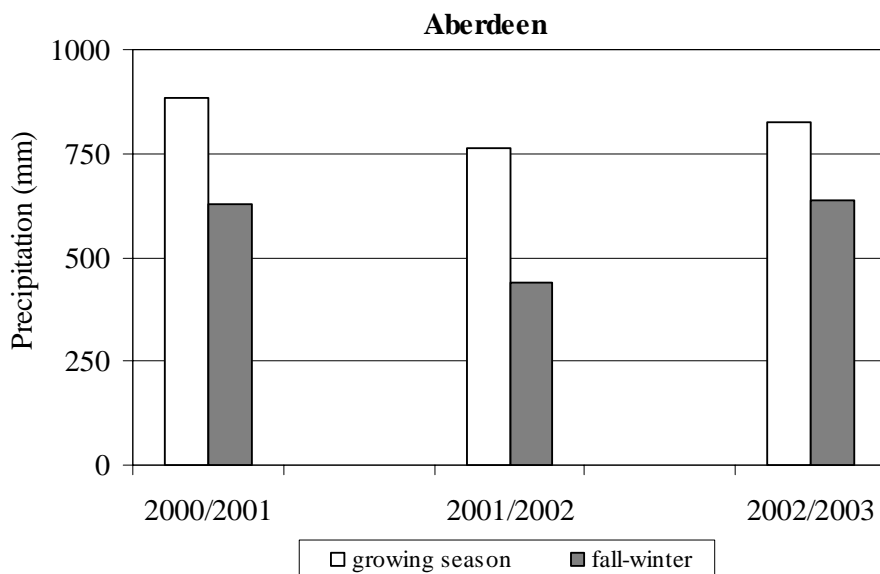


Figure 2.2: Precipitation (mm) during the growing season of winter oilseed rape and over fall-winter in Aberdeen.

Table 2.3: Description of soil parameters in Aberdeen.

Soil	Method	Reference	Unit	Year 1	Year 2	Year 3
pH	CaCl ₂	Schofield and Taylor, 1955	-	6.0	6.1	6.6
Organic matter	LOI	Schulte and Hopkins, 1996	%	10.8	10.2	8.9
P	Morgan	MAFF, 1986	mg kg ⁻¹	5.7	6.2	16.1
SO₄-S	CaH ₄ (PO ₄) ₂	Gay, 2004	mg kg ⁻¹	13.5	18.5	10.4
K	Morgan	MAFF, 1986	mg kg ⁻¹	158	168	211
Mg	Morgan	MAFF, 1986	mg kg ⁻¹	80	71.7	270

note: data delivered by the SAC, Scotland

Inverness

Inverness (W 4° 5', N 57° 53'), known as the capital of the Scottish Highlands, is located on the northeast coast of Scotland. The experimental site has a maritime climate. Meteorological records were collected during the course of investigations on the local meteorological site at Kinloss and are given in the appendix (Table A.3). During experimentations, the mean temperature was about 8.5°C, the sum of sun hours about 1240 h and the rainfall varied between 518 mm and 726 mm (Figure 2.3). The long-term mean annual precipitation for Inverness is 640 mm, which is comparable to that in Braunschweig (see above). In all three experimentation

seasons about 50% of precipitation fell over the period from fall to winter (Figure 2.3). Sulphate losses by leaching were presumably highest in the second experimentation season with precipitation rates of 726 mm during the growing season (September-July) and 454 mm over fall-winter (September-February) (Figure 2.3).

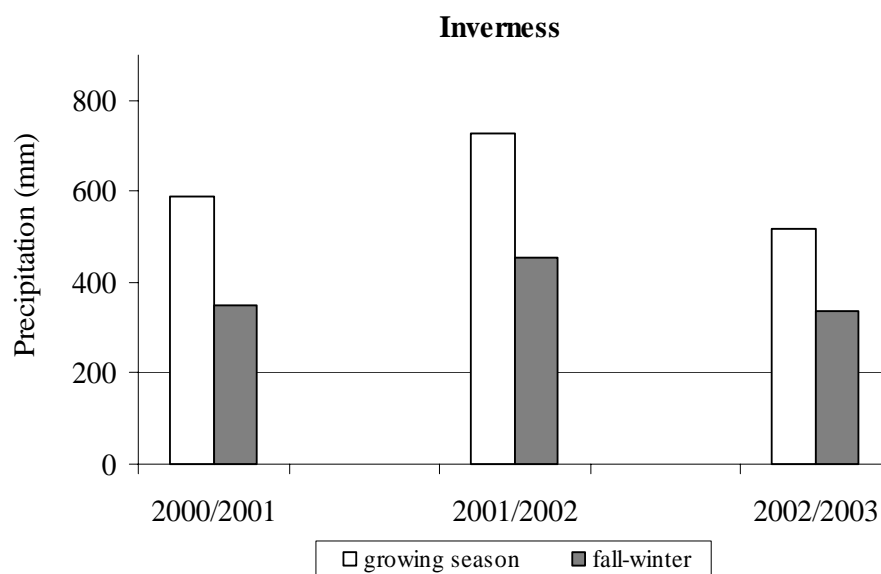


Figure 2.3: Precipitation (mm) during the growing season of winter oilseed rape and over fall-winter in Inverness.

The soil type in Inverness is a Cambisol with a mean soil pH of 5.6. Chemical soil characteristics are summarised in Table 2.4. The soil can be described as a loamy sand soil with a high organic matter content and a sufficient nutrient supply (Table 2.4).

Table 2.4: Description of soil parameters in Inverness.

Soil parameters	Method	Reference	Unit	Year 1	Year 2	Year 3
pH	CaCl ₂	Schofield and Taylor, 1955	-	5.5	5.9	5.3
Organic matter	LOI	Schulte and Hopkins, 1996	%	8.2	6.9	5.9
P	Morgan	MAFF, 1986	mg kg ⁻¹	6.7	7.4	10.6
SO₄-S	CaH ₄ (PO ₄) ₂	Gay, 2004	mg kg ⁻¹	14.9	8.8	21.8
K	Morgan	MAFF, 1986	mg kg ⁻¹	55	55.1	113
Mg	Morgan	MAFF, 1986	mg kg ⁻¹	76.9	88.5	41.2

note: data delivered by the SAC, Scotland

2.2 Experimental design

Field experiments were conducted on three sites during three consecutive seasons to investigate the influence of fungal infections, S and N fertilisation, cultivar and fungicide treatment on mineral composition and S-containing primary and secondary metabolites that are supposed to be essential for *SIR* (Table 2.5).

Table 2.5: Design of field experiments in Braunschweig and Scotland and occurrence of *Pyrenopeziza brassicae* and *Leptosphaeria maculans* infections.

Site	<i>Braunschweig</i>	<i>Aberdeen</i>	<i>Inverness</i>
Variety	<i>Bristol/Lipton</i>	<i>Bristol/Lipton</i>	<i>Bristol/Lipton</i>
S rate [kg ha⁻¹]	0/150	0/100	0/100
N rate [kg ha⁻¹]	100/200	100/200	100/200
Fungicide	-/+	-/+	-/+
Infections by <i>P. brassicae</i>	2001: no 2002: yes 2003: no	yes yes yes	yes yes yes
Infections by <i>L. maculans</i>	2001: no 2002: yes 2003: no	yes yes yes	no no no

Field trials were conducted in plots of 40-60 m² and each treatment had four replicates. In Braunschweig, the plots were arranged in a completely randomised block design with four blocks of 16 experimental units. Cultivars, S, N and fungicide treatments were completely randomised within each block according to Schuster and von Lochow (1979). In Scotland, the experiment was a split-plot design in four blocks. The fungicide treatment was randomised in two main plots in each block and cultivars, S and N rates were randomised in eight sub-plots in each main plot. Maps of the experimental design in Braunschweig and Scotland are shown in the appendix (Table A.4 and A.5).

The trials were sown in August/September with a seed density of 6 kg seeds ha⁻¹ (individual data given in the appendix: Table A.6 to A.8). Growth stages (G.S.) of winter oilseed rape were established by using the BBCH scale of Strauss et al. (1994). Two oilseed rape cultivars with different susceptibilities to *P. brassicae* and *L. maculans* were grown: *Bristol*, susceptible to

P. brassicae and resistant to *L. maculans* (Gladders et al., 1998) and *Lipton*, resistant to *P. brassicae* and susceptible to *L. maculans* (HGCA Recommended List WOSR 2003) (Table 2.5).

S was applied as potassium sulphate (K_2SO_4) to the soil and potassium (K) was balanced by fertilising adequate amounts of potassium chloride (KCl) to the control plots. The S application rates were of 0 and 150 kg S ha⁻¹ in Braunschweig and of 0 and 100 kg S ha⁻¹ in Scotland (Table 2.5).

In Braunschweig, the infection pressure for *P. brassicae* is usually extremely low and in order to promote infections by this pathogen in fall 2000 no S was applied. In the first year of experimentation (2000/2001), uniformly 75 kg S ha⁻¹ were applied two times in spring in dependence on precipitation and growth development of the crop (see Appendix: Table A.6). Since in the next two experimental seasons *P. brassicae* was introduced in the field (see later on), S was applied in fall as well. In the second season (2001/2002), S was applied at rates and doses of two times 40 kg S ha⁻¹ in fall and one time 70 kg S ha⁻¹ in spring (see Appendix: Table A.6). In the third season (2002/2003), S was split as follows: 70 kg S ha⁻¹ in fall (G.S. 08) and 80 kg S ha⁻¹ in spring (G.S. 19) (see Appendix: Table A.6).

In Scotland, S was applied in one dose (G.S. 19-30) at both sites in the first season (2000/2001) (see Appendix: Table A.7 and A.8). In the next two seasons, S was split in two doses in autumn (G.S. 02-08) and in spring (G.S. 14-19) (see Appendix: Table A.7 and A.8).

N was applied as ammonium nitrate (NH_4NO_3) at rates of 100 and 200 kg N ha⁻¹ (Table 2.5). The N dose was split in two equal doses in Braunschweig according to the developmental stage of the crop (individual data given in the appendix: Table A.6). In Scotland, 100 kg N ha⁻¹ were applied to all plots at the start of the vegetation period (G.S. 14-15) and an additional 100 kg N ha⁻¹ were applied at the beginning of stem elongation in dependence on the treatment (G.S. 19-30) (individual data given in the appendix: Table A.7 and A.8).

For the weed control clomazone was used immediately after planting (individual data given in the appendix: Table A.6 to A.8).

S-free fungicides were applied to all experimental sites with the active ingredients carbendazim plus flusilazole, vinclozolin, iprodion and tebuconazol. Flusilazole (250 g L⁻¹) plus carbendazim (125 g L⁻¹) is highly effective against *P. brassicae*, vinclozolin is effective against *S. sclerotiorum*, iprodion is effective against *S. sclerotiorum*, *A. brassicae* and *Botrytis cinerea*

(grey mold) and tebuconazol is highly effective against *L. maculans* and effective against *P. brassicae*, *S. sclerotiorum* and *A. brassicae*. In the plus fungicide treatment of the experimentation the plots received all of these fungicides, while in the minus fungicide treatment no flusilazole (250 g L⁻¹) plus carbendazim (125 g L⁻¹) was applied (Table 2.5).

Flusilazole plus carbendazim was applied in Braunschweig to the plus fungicide plots in fall at rates of 1 L ha⁻¹ (individual data given in the appendix: Table A.6). At this experimental site all plots received additionally 1.5 kg ha⁻¹ vinclozolin and 3 L ha⁻¹ iprodion at the beginning of flowering (G.S. 60-63) (see Appendix: Table A.6). In Scotland, the treated plots received 0.4 L ha⁻¹ flusilazole plus carbendazim in fall (G.S. 14-15) and in spring (G.S. 19-30), respectively (individual data given in the appendix: Table A.7 and A.8). In Aberdeen, severe weather conditions in fall-winter 2000/2001 and subsequent Foot & Mouth outbreak made changes in the fungicide programme necessary: 0.4 L ha⁻¹ flusilazole plus carbendazim was applied in April (G.S. 30) and 0.5 L ha⁻¹ tebuconazol was applied in May (G.S. 60-63) (see Appendix: Table A.7).

Pyrenopeziza brassicae epidemics in oilseed rape crop are generally very low in Braunschweig. Even without S fertilisation in fall in the first year of experimentation no natural infection by *P. brassicae* was observed (see Table 2.5). In order to promote infections by this fungal pathogen, in fall 2001 and 2002 *P. brassicae* was introduced in the field by scattering infected straw from Scotland on the plots soon after sowing. Thus, in the second year of experimentation (2001/2002), infections of oilseed rape by *P. brassicae* were observed (Table 2.5). In this season, beside infections by *P. brassicae*, *L. maculans* infected also the oilseed rape crop (Table 2.5). Symptoms of the later pathogen were visible in autumn, while in spring symptoms of *P. brassicae* were dominating. Symptoms of infection by *P. brassicae* and *L. maculans* are illustrated in Figure 2.4.

In Scotland, over three years research period, the infection rate and severity of *P. brassicae* in winter oilseed rape crop ranged from moderate to high (see Appendix: Table A.19 to A.21). Beside infections by *P. brassicae*, additional infections by *L. maculans* (Figure 2.4), *P. parasitica* (see Appendix: Table A.19 and A.20), *A. brassicae*, *S. sclerotiorum* and *B. cinerea* were also found.

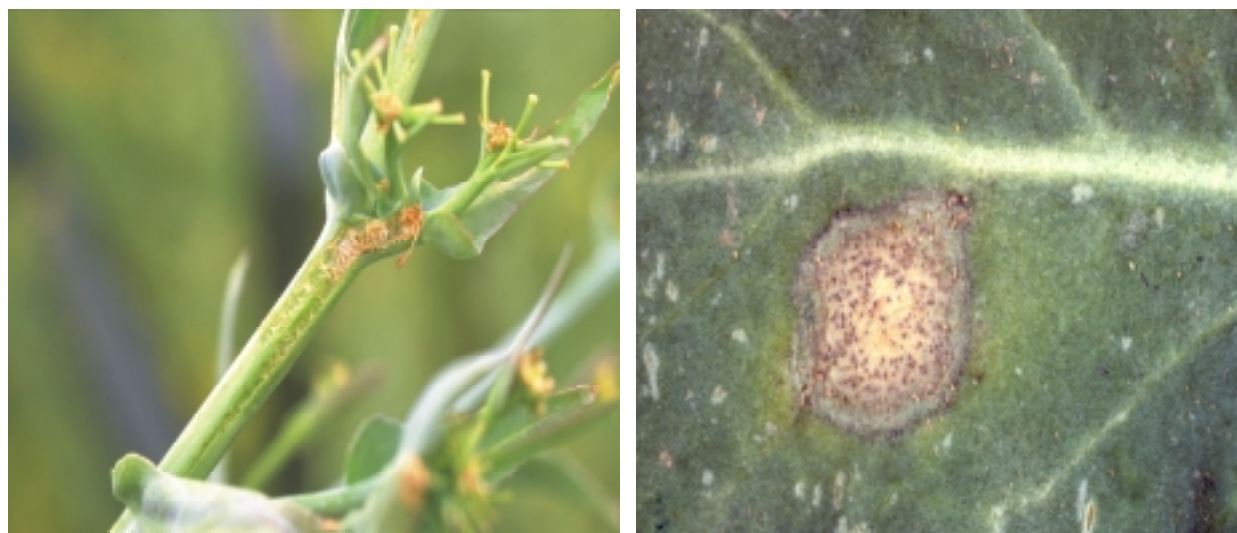


Figure 2.4: Symptoms of infection by *Pyrenopeziza brassicae* (left) and *Leptosphaeria maculans* (right) in *Brassica napus* crop (photo left: E Schnug; photo right: SAC).

2.3 Sampling procedures

Leaf samples were collected at the beginning of stem elongation (G.S. 50-53), and seed, straw and pod wall samples were taken at harvest (G.S. 97-99). Sampling and combine harvesting have been carried out in the centre of the plots in order to eliminate side effects.

Younger, fully expanded leaves were collected because S induced resistance responses are supposedly highest within growth stages of high metabolic activity (Schnug and Haneklaus, 1998). Beside whole leaf samples, leaf disc samples (16 mm diameter) were taken in Braunschweig (2001/2002) and Aberdeen (2001/2002 and 2002/2003) from visually infected and visually non-infected leaves by the fungus *P. brassicae* at the beginning of stem elongation (G.S. 50-53), too.

2.4 Plant analysis

Whole leaf samples were split and either shock frozen in liquid nitrogen and finally freeze-dried or dried with fresh air in a ventilated oven at 60°C until the constancy of weight. Leaf disc samples were shock frozen in liquid nitrogen and then freeze-dried. Straw, pod wall and seed samples were oven-dried with fresh air at 60°C and 30°C, respectively until the constancy of weight. Oven-dried plant samples were fine-ground to a particle size < 0.12 mm using a *Retsch* ultra-centrifugal mill prior to analysis. Freeze-dried whole leaf and leaf disc samples and seed samples were fine-ground using a mortar or a coffee mill for further analysis.

2.4.1 Mineral nutrients

Total N content was determined using a standard micro-Kjeldahl method (Schlichting and Blume, 1966).

Total S: For the determination of the total S content by X-ray fluorescence spectroscopy (X-RF), tablets were prepared by mixing 1.1 g of fine-ground leaf material with 4.4 g of Hoechst wax 'C'. The analysis was carried out according to Schnug and Haneklaus (1999). The total S content in seeds was determined by microwave digestion: ground seed material was digested for 20 minutes in a microwave (S1200 mega) using a mixture of HNO₃ (65%) and H₂O₂ (35%) (4:1, vv). After cooling, samples were diluted with bi-distilled water to a final volume of 25 ml and afterwards filtered. The analysis was carried out by inductively coupled plasma-atomic emission spectroscopy (ICP-AES). Beside total S content, the concentration of K, Ca, Mg, P, Si, Cl, Na was also determined in leaf and seed material by X-RF and ICP-AES, respectively.

Alternatively, the total S content in seeds was calculated from the seed total GSL content by employing the conversion algorithm derived from the X-RF method. Based upon a good correlation between total S and total GSL content in the seed (Schnug and Haneklaus, 1988), the total GSL content in seeds can be indirectly calculated from the total S content derived from the X-RF analysis and vice-versa, the total S content in seeds can be calculated from the total GSL content derived from the HPLC analysis. The conversion algorithm is (according to X-RF calibration with reference standards; non-linear calibration) (Schnug and Haneklaus, 1990):

$$y = -5.6 x + 2.8 x^2 - 0.12 x^3 + 3.5$$

where: y = total GSL concentration ($\mu\text{mol g}^{-1}$)

x = total S concentration (mg g^{-1}).

Sulphate (SO_4^{2-}) was determined by extracting the plant material according to Novozamsky et al. (1986). The extraction was carried out with bi-distilled water for 30 min using a lab-shaker. After the filtration of plant debris, sulphate was precipitated as barium sulphate (BaSO_4) using barium chloride (BaCl_2). The precipitate was then separated by centrifugation, washed with bi-distilled water and dissolved in $(\text{NH}_4)_4$ -EDTA (Novozamsky et al., 1986). The $(\text{NH}_4)_4$ -EDTA extract was used for sulphate determination either by ICP-AES or ion chromatography (IC). For IC determination, the $(\text{NH}_4)_4$ -EDTA extract was further diluted with bi-distilled water in a ratio of 1:4. The sulphate-sulphur ($\text{SO}_4\text{-S}$) fraction was calculated from the sulphate content.

2.4.2 Organic sulphur compounds

Protein S was calculated as the difference between total S content and S bound in free cysteine, GSH, GSLs and SO_4^{2-} .

Free cysteine, γ -glutamyl-cysteine and *glutathione (GSH)* were determined by HPLC according to Hell and Bergmann (1990). The extraction was carried out using 20-30 mg fine ground freeze-dried leaf material and 1 ml of 0.1 M HCl containing 4% PVP (Polyvidon-25). After the removal of plant debris by subsequent centrifugation, the supernatant was used for reduction in the dark with dithiothreitol (DTT). The assay contained: 1M Tris/HCl, pH 8, 10 mM DTT, 0.08 M NaOH, H_2O , plant extract (supernatant) and standards. After 1 h reduction by DTT, the sulphhydryl groups were derivated with 25 μl of 10 mM bromobimane (Sigma No. B-4380) and then stabilised by the addition of 705 μl acetic acid (5%). The separation of cysteine, γ -glutamyl-cysteine and GSH was carried out by HPLC using a 250 x 4.6 mm Nova-Pak C_{18} column (4 μm) (Water 044380). Detection was conducted at 480 nm in a fluorescence detector. The chromatograms in conjunction with fluorescence spectra were used for the identification of these metabolites and calibration standards for their quantification.

Glucosinolates (GSLs) in leaf and seed samples were determined according to Rosa (1992) and Anon (1990b), respectively. Glucotropaeolin and sinigrin were used as internal standards for leaf and seed samples at the start of the extraction procedure. The extraction of GSLs was carried out using boiling methanol/water (70:30) and finally plant debris was removed by centrifugation. In the case of seeds, a further step for the removal of proteins was necessary. Proteins were precipitated using lead-barium acetate and then the precipitate was removed by centrifugation. Glucosinolates were obtained as desulphoglucosinolates after loading the methanol extracts on a DEAE anion-exchange column (DEAE-Sephadex A-25), followed by sulphatase treatment and elution with HPLC water. Individual desulphoglucosinolates were separated by HPLC using a 250 x 4.6 mm HyPersil C_{18} column (5 μm) (Phenomenex 00G-0152-E0) and elution gradient with acetonitrile. Detection was realised by measuring the absorbance at 229 nm in an UV detector. The chromatograms in conjunction with diode array UV spectra and BCR standards were used for the identification and quantification of individual GSLs.

Individual GSL concentrations were calculated according to the following formula:

$$\mu\text{mol of GSL g}^{-1} \text{ DM} = \frac{A_{\text{GSL}} * \text{RF} * C_{\text{is}}}{A_{\text{is}} * \text{SW}}$$

where: $\mu\text{mol of GSL g}^{-1} \text{ DM}$ = glucosinolate content (μmol) in 1 g of dry matter

A_{GSL} = glucosinolate area

RF = response factor (for RF values see Rosa, 1992)

C_{is} = internal standard concentration (μmol)

A_{is} = internal standard area (glucotropaeolin/sinigrin)

SW = sample weight (g).

The total GSL content was obtained by summing up individual GSLs.

L-cysteindesulphhydrase (LCD) and O-acetylserine(thiol)lyase (OAS-TL) activities

Enzyme activities (LCD and OAS-TL) were determined by the Institute of Botany, University of Hannover, Germany. The research group in Hannover is also a member of the German Plant S Group (DFG-Forschergruppe 383; Teilprojekt 7 – Schmidt/Papenbrock) and results between working groups were exchanged in mutual agreement.

For analysis, freeze-dried and ground plant material (leaf discs) was suspended in cold 20 mM Tris/HCl, pH 8 and centrifuged to remove cell debris. The supernatant was used in the enzyme assays. The *LCD activity* was measured by the release of sulphide from cysteine (Siegel, 1965). The assay contained: 0.8 M L-cysteine, 2.5 mM DTT, 100 mM Tris/HCl, pH 9 and enzyme extract. The formation of methylene blue was determined at 670 nm in a spectral photometer and quantified using a Na_2S standard curve. The *OAS-TL activity* measurement was based on cysteine concentration determination (Gaitonde, 1967). The assay contained: 5 mM OAS, 5 mM Na_2S , 33.4 mM DTT, 100 mM Tris/HCl, pH 7.5 and enzyme extract (Schmidt, 1990). The cysteine concentration was determined at 560 nm in a microplate reader (Fluostar Optima, BMG Labtechnologies, Offenburg) and quantified using a L-cysteine standard curve. For a complete description of procedures of enzyme activity determinations see Riemenschneider (2003).

2.5 Disease assessment

Disease assessment data from the experimental fields in Scotland were supplied by the Scottish Agricultural College (SAC), Aberdeen, Scotland.

In Scotland, the development of *P. brassicae* in winter oilseed rape crop was observed during all years of experimentation (see Table 2.5). Infections were scored regularly during the whole growth period. Besides scoring of infections caused by *P. brassicae*, plants were also scored for infections caused by other fungal pathogens such as *L. maculans*, *A. brassicae*, *P. parasitica*, *S. sclerotiorum*, *B. cinerea*.

Since visible symptoms of *P. brassicae*, such as white spore masses that erupt through the leaf surface, do usually not occur before February/March and are also difficult to identify under field conditions over winter period (Fitt et al., 1998), for scoring of *P. brassicae* infections in earlier growth stages it was necessary to incubate plant samples. Therefore, over fall-winter, samples were taken every 1 to 2 weeks (except the first season; weather conditions not permitting) by randomly choosing 10 plants from untreated plots (minus fungicide plots; see chapter 2.2). After incubating them in a damp chamber over night, the disease incidence and severity of fungal pathogens were assessed. When symptoms of *P. brassicae* were visible in the field, the scoring of fungal infections was carried out visually in all plots at monthly intervals.

The disease incidence (% plants infected) and severity (% leaf, stem or pod area infected) were calculated according to the following formulae (Sutherland et al., 2004):

$$\text{disease incidence (\%)} = \frac{\text{plants infected}}{\text{total number of plants}} \times 100$$

$$\text{disease severity (\%)} = \frac{\text{leaf (stem, pod) area infected}}{\text{total leaf (stem, pod) area}} \times 100$$

In Braunschweig, infections of winter oilseed rape crop by *P. brassicae* and *L. maculans* were detected in the season 2001/2002 (see Table 2.5). *Leptosphaeria maculans* symptoms (see Figure 2.4) were only visible in fall. The scoring of *L. maculans* infection severity was carried out visually in all plots in fall 2001 (G.S. 14-15) by counting the number of *L. maculans* lesions on leaves in a 0.5 x 1 m frame. The infection severity was scored as follows: 1-2 = low infection rate with 1-2 spots/leaf; 3-5 = medium infection rate with 3-5 spots/leaf; 6 = high infection rate with > 6 spots/leaf. For each plot in total 5 separate counts were made and mean score values were determined. In spring 2002, symptoms of *P. brassicae* were visible in the field (see Figure

2.4), but a scoring of infection was not possible because of the high inconsistency of the infection rate and severity within individual plots.

2.6 Statistical analysis

2.6.1 Standard statistical analysis

For the statistical analysis the SPSS software package version 10 was employed (SPSS, 1999). In the present work, the GLM multivariate procedure and correlation analysis were carried out.

The GLM multivariate procedure was employed to assess the influence of the treatments on individual parameters. For all three experimental sites cultivar, S, N and fungicide were tested as fixed factors. The experimental design with four factors allows the investigation of interactions of fourth degree, which however is difficult to describe and explain. In this research work, only the main effect of the sources of variation (factors) is statistically explored. Multivariate exploitation of the data and the significance of site and year of experimentation for plant parameters will be provided in an extra publication. Three factorial ANOVA was carried out to determine the effect of S, cultivar and dates of assessment of scoring values on the level of fungal infections. One factorial ANOVA was employed in the case of leaf discs in order to establish the influence of infections by *P. brassicae* on the cysteine and GSH content and on the LCD and OAS-TL activity. The Student-Newman-Keuls test was used to determine which means were significantly different from each other at the 5% significance level (LSD_{5%}).

The correlation analysis (Pearson Correlation, two-tailed) was carried out in order to identify relationships between variables.

The outlier test was used to determine if measured values were reliable (Schnug, 1985). Values characterised as outliers by employing this test were not used in statistics and in the appendix are termed as missing values (m.v.). If some samples for different treatments were not available (i.e. > 12 samples), the whole data set was not statistically analysed.

Significance levels obtained from standard statistics were coded as follows:

n.s. not significant;

* significant, $p < 0.05$;

** highly significant, $p < 0.01$;

*** very highly significant, $p < 0.001$.

2.6.2 Geostatistical analysis

The fundamental difference between standard statistics and geostatistics is that the later is based on the theory of regionalised variables, i.e. variables that have a spatial distribution (Matheron, 1971; Journel and Huijbredgts, 1997). Geostatistics (variography) describe the spatial relationship between the measurements of variables at different sampling locations (Matheron 1971, Journel & Huijbredgts 1997). Basic information about geostatistics are provided by Isaaks and Srivastava (1989), Journel (1989) and Gassner and Schnug (2003).

For the present work, geostatistics were employed to estimate the probability distribution of *L. maculans* infections and to assess the spatial variability of plant parameters (i.e. total S content, GSH and GSL concentrations) in Braunschweig (season 2001/2002). For each plot (3 x 20 m) coordinates of the centre point were taken and the determined values for individual parameters assigned. The geometric centre point of each plot was further used for geocoding.

Spatial autocorrelations for different plant parameters and *L. maculans* infection severity were analysed using semi-variogram analysis. The Variowin software package version 2.2 was employed (Pannatier, 1996). Procedures to develop experimental variograms and to fit model variograms are given by Cressie (1993), Isaaks and Srivastava (1989) and Journel (1989).

The semi-variogram provides a measure of spatial correlation by describing how sample data are related in dependence on distance and direction (Isaaks and Srivastava 1989). An example of a spherical semi-variogram model is given in Figure 2.5. The average squared difference between paired data values ($\gamma(h)$) is plotted against h , the distance or lag separating the pairs (Figure 2.5). The shape of this plot summarises the type of spatial structure or dependence and the distance over which this dependence occurs. If there is spatial dependence among the data, $\gamma(h)$ typically increases with the separation distance h , may level off and even decrease after certain distance. The value of $\gamma(h)$ when the semi-variogram levels off is called the sill ($C_0 + C_1$) and represents the distance between points above which autocorrelation no longer exist. The distance h at which the sill is attained is called the range (a) and represents the maximum distance at which there is spatial continuity. The semi-variogram at zero (lag $\gamma(0)$) must be zero, but in reality the extrapolated semi-variogram usually intercepts the ordinate at a positive value known as the nugget variance (C_0). The nugget effect can rise from several factors including sampling error and spatially dependent variation occurring over distances smaller than the sampling interval.

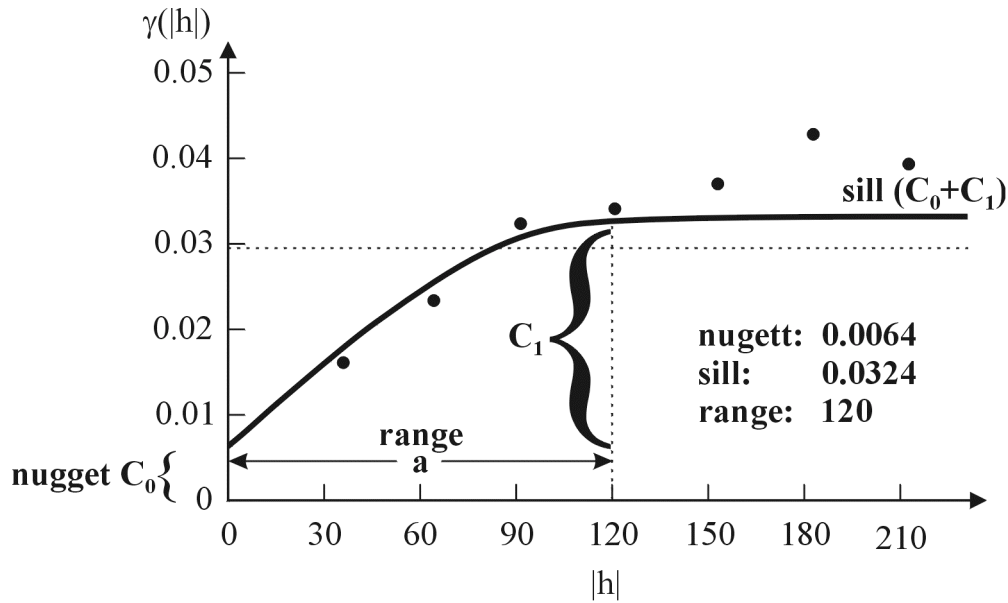


Figure 2.5: Example for a spherical semi-variogram model.

A quantification of the contribution of random variation to the semi-variance observed in a data set can be estimated from the ratio of nugget semi-variance to sill semi-variance (NR) (Trangmar et al., 1985):

$$NR = \dots \frac{C_0}{C_0 + C_1} * 100$$

This ratio was used to describe the strength of the spatial dependence within the field (Cambardella et al., 1994), where:

- NR \leq 25 % : indicates a strong spatial dependency
- 25 % < NR < 75 % : indicates a moderate spatial dependency
- NR \geq 75 % : indicates a weak spatial dependency.

The semi-variogram model information was further used to create spatial distribution maps using the kriging algorithm of the Surfer software package (Keckler 1995). Kriging is a method of optimal unbiased estimation of variables between sampling locations based on parameters of the semi-variogram and initial data values (Clark, 1979). Predictions are weighted averages of the actual measurements, with values for the weights derived from the solution of a set of equations determined by the semi-variogram and the location and orientation of the sample points relative to each other and to the point or area being predicted.

For geo-referenced data that showed no autocorrelation, interpolation maps were created using the inverse distance procedure. Inverse distance weighting do not use the geostatistical properties of the data to optimise the interpolation method. Instead they depend on a user-defined weighting parameter (Roberts et al., 1993). These interpolative methods are simpler than kriging, but require some previous knowledge about the phenomena being mapped to choose an appropriate weighting value (Roberts et al., 1993).

For the infection severity of *L. maculans*, which is a descriptive, categorical variable, a probability map was created using the so-called indicator kriging procedure (Journel, 1983). To characterise the probability distribution of *L. maculans* infections, each observed score value $z(x)$ was transformed to an indicator datum $I(x, \text{score})$ according to Marinoni (2003):

$$I(x, \text{score}) = \begin{cases} 1, & \text{if } z(x) = \text{score} \\ \dots\dots\dots & \\ 0, & \text{otherwise} \end{cases}$$

where $I(x, \text{score})$ is the transformed indicator at location x for a specified score value. Following the above transformation, measured values for infection severity were transformed to indicator variables by scoring them either as 1 or 0. At every location x where the observed fungal infection severity $z(x)$ had a specified score value of 6, an indicator value of 1 was assigned, which is equivalent to 100% risk of severe infections. All other score values received a value of 0 (0% probability of severe infections). The spatial continuity of the transformed score values was modelled with variography and the resulting model was used to make a kriged interpolation surface.

3 Results

Earlier reports revealed that S fertilisation has a positive effect on the concentration of S-containing metabolites in plant tissues (De Kok et al., 1981; Schnug et al., 1995b; Schnug, 1997; Haneklaus et al., 1999; Wielebski et al., 1999). Cysteine, GSH, GSLs and H₂S were reported to play an important role in the plant defence system against pathogens (Schnug and Ceynowa, 1990; Burandt et al., 2001; Gullner and Kömives, 2001). However, there are still open questions regarding the efficacy of these S-containing compounds in pathogenesis as well as regarding the impact of the S nutritional status on plant S fractions, particularly under field conditions. It is therefore the main aim of this research work to identify the S-containing metabolites involved in the resistance of oilseed rape against fungal pathogens in dependence on the S supply under natural conditions.

In the first two chapters, the influence of S and N fertilisation, cultivar and fungicide treatment on different S fractions and on crop productivity is presented (chapter 3.1 and 3.2). In the third chapter, the results of field experiments conducted in Scotland investigating the potential of S fertilisation to reduce fungal infections are shown (chapter 3.3). In the last two chapters, the changes in metabolite concentrations and enzymatic activities in response to fungal infections and relationships between fungal infections and S-containing metabolites are studied (chapter 3.4 and 3.5). Because of the high spatio-temporal variability of plant and environmental factors, the identification of mechanisms involved in *SIR* is generally more difficult under field conditions. Therefore, infection-directed sampling strategies (i.e. leaf discs sampling) (chapter 3.4) and geostatistics (chapter 3.5) were employed in order to identify metabolites and processes that entail *SIR*.

3.1 Influence of sulphur and nitrogen fertilisation, cultivar and fungicide treatment on mineral composition and yield of winter oilseed rape

Previous results indicated that a sufficient S nutrition is important for oilseed rape, with respect to yield and crop quality, because of its high S demand (Gupta et al., 1997; Pedersen et al., 1998; Haneklaus and Bloem, 2000). An interaction between S and N has been also noted, indicating that there is a requirement to balance the application of these nutrients for an optimum response (Schnug et al., 1993a). Although some data on the mineral composition of oilseed rape in dependence on the S and N supply are available from field experiments (Donald et al., 1993; Schnug and Haneklaus, 1993b), only a few data are available from oilseed rape plants grown on sites characterised by different S status. It is therefore the aim of this part of the research work to

assess the influence of the S and N supply as well as the influence of the cultivar and fungicide treatment on mineral composition and yield of field-grown oilseed rape plants at three different locations, i.e. Braunschweig, Aberdeen and Inverness. The statistical analysis of data was carried out by employing the multivariate GLM procedure. In this chapter, the influence of the treatments on the total S and total N content, sulphate-S levels and seed yield is presented for each study site and experimental season. Individual data for all mineral nutrients checked are given in the appendix (Table A.9 to A.12).

Dependence of mineral composition and seed yield on the S supply

The effect of S fertilisation on the total S content in young fully developed leaves and seeds of winter oilseed rape during three years research period is summarised in Table 3.1. The analysis of variance showed that differences between control and S fertilised plots were significant ($p < 0.001$) for both leaves and seeds (Table 3.1). The total S content in young leaves at the start of stem elongation increased on average by 2.1-fold, 1.5-fold and 1.7-fold in Braunschweig, Aberdeen and Inverness, respectively when S was applied to the soil (Table 3.1). Comparing the experimental seasons, it can be seen that the total S content in the leaf tissue varied largely (Table 3.1). This indicates the large variability of the soil S supply.

In the control plots, the total S content in young leaves varied from 2.9 mg S g⁻¹ to 6.2 mg S g⁻¹ (Table 3.1) and was in the range between severe and latent S deficiency (Schnug and Haneklaus, 1998). Therefore, the total S content in plants from these plots was below the value for a maximum yield. On the other hand, S fertilised plots were sufficiently supplied with S and concentrations of > 6.5 mg S g⁻¹ were found (exception third season) (Table 3.1) (Schnug and Haneklaus, 1998).

In the case of seeds, the mean increase of the total S content as a result of S fertilisation was 1.2-fold in Braunschweig and Inverness, and 1.1-fold in Aberdeen (Table 3.1). Comparing the experimental sites, it can be noted that in Braunschweig the total S content in seeds from control plots (3.2 mg S g⁻¹) was higher than in Aberdeen (2.9 mg S g⁻¹) and Inverness (2.5 mg S g⁻¹) (mean over three seasons) (Table 3.1). Taking into account that these three sites differ in the amount of S depositions (see Table 2.1), these findings might suggest the contribution of atmospheric S to the plant supply. Seasonal variations of the total S content in seeds were not as large as observed in the case of leaves (Table 3.1).

Table 3.1: Effect of S fertilisation on the total S content in young fully developed leaves and seeds of winter oilseed rape and on seed yield during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	Total S						Seed yield		
	$mg\ g^{-1}$						$t\ ha^{-1}$ (8% moisture)		
	Leaves			Seeds			BS	ABR	INV
	BS	ABR	INV	BS	ABR	INV			
2000/2001									
S₀	3.98	6.22	3.66	3.20	2.76	2.29	3.34	3.58	2.42
S₁	10.1	9.06	6.66	3.58	2.98	2.87	3.59	3.61	2.86
LSD_{5%}	0.44	0.58	*	0.09	0.08	0.07	0.21	0.12	0.17
2001/2002									
S₀	4.78	4.18	3.46	3.37	3.02	2.29	1.75	2.97	2.30
S₁	9.29	7.60	7.58	4.10	3.36	3.08	1.88	3.15	2.37
LSD_{5%}	0.35	0.40	0.35	0.16	0.11	0.07	0.27	0.21	0.16
2002/2003									
S₀	2.95	4.87	4.41	2.88	2.90	3.04	2.95	3.53	2.77
S₁	5.55	6.08	5.39	3.65	3.16	3.37	2.94	3.53	2.84
LSD_{5%}	0.34	0.17	0.27	0.08	0.08	0.10	0.11	0.16	0.38
note: * not statistically analysed									

Since S deficiency was observed on all three experimental sites (see above), it was investigated if the seed yield increased significantly in response to S applications. The effect of S fertilisation on seed yield (at 8% moisture) is shown in Table 3.1. The results from the GLM procedure revealed that S had no significant effect on the final seed yield and only a slight yield increase was observed when S was applied to the soil (Table 3.1). The seed yield increased on average by 1.1-fold in Braunschweig and Inverness and almost no increase was found in Aberdeen (Table 3.1). This implies that other factors were yield limiting. For instance, in the third season in Aberdeen, young leaves were potassium (K) deficient (see Appendix: Table A.10) and thus, the uptake of S might have not been efficient in this season. Additionally, oilseed rape plants were highly attacked by fungal pathogens during the experimentation (see chapter 3.3, 3.4 and 3.5 and Appendix: Table A.18 to A.21).

The seed yields on all three experimental sites were poor and over three years ranged from 1.8 t ha⁻¹ to 3.6 t ha⁻¹ in Braunschweig, from 3.0 t ha⁻¹ to 3.6 t ha⁻¹ in Aberdeen, and from 2.3 t ha⁻¹ to 2.9 t ha⁻¹ in Inverness (Table 3.1). The poor yields might be due to the presence of several abiotic and biotic factors that are influencing the crop growth and development.

Site S status (i.e. atmospheric S depositions) probably had some influence on yield. The mean yield at the low S site in Aberdeen was 3.4 t ha⁻¹ compared to 2.5 t ha⁻¹ at the very low S site in Inverness (Table 3.1).

At the experimental site in Braunschweig, straw and pod wall samples were also investigated for the content of mineral elements. The effect of S fertilisation on the total S content in straw and pod walls is shown in Table 3.2.

Table 3.2: Effect of S fertilisation on the total S content in straw and pod walls of winter oilseed rape during three years (2000-2003) in Braunschweig.

Treatment	<i>Total S</i>	
	<i>mg g⁻¹</i>	
	<i>Straw</i>	<i>Pod walls</i>
<i>2000/2001</i>		
S₀	4.82	n.a.
S₁	4.95	n.a.
LSD_{5%}	0.21	n.a.
<i>2001/2002</i>		
S₀	1.69	1.16
S₁	2.19	1.38
LSD_{5%}	0.22	0.07
<i>2002/2003</i>		
S₀	2.38	4.21
S₁	5.51	10.2
LSD_{5%}	0.42	0.80
note: n.a. – not available		

The analysis of variance showed that S fertilisation had a significant influence ($p < 0.001$) on the total S content in both straw and pod walls (exception first season) (Table 3.2). During experimentations, an increase from 1.2-fold to 2.4-fold was found in response to S. Seasonal differences with respect to the total S content in straw and pod walls were observed as well (Table 3.2).

Sulphate is known to represent a large fraction of the total S in leaves (Blake-Kalff et al., 1998). Since S fertilisation has proved to increase significantly the total S content in the leaf tissue (see Table 3.1), it was also investigated if there was a significant effect of S applications on the SO_4 -S content. The effect of S fertilisation on the SO_4 -S content in young fully developed leaves of winter oilseed rape at the start of stem elongation is summarised in Table 3.3.

Table 3.3: Effect of S fertilisation on the SO_4 -S content in young fully developed leaves of winter oilseed rape at the start of stem elongation during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	SO_4 -S		
	$mg\ g^{-1}$		
	BS	ABR	INV
2000/2001			
S₀	0.80	3.35	0.79
S₁	5.55	6.29	3.51
LSD_{5%}	0.61	*	0.53
2001/2002			
S₀	0.95	0.68	0.29
S₁	2.43	1.26	1.34
LSD_{5%}	0.45	0.21	0.21
2002/2003			
S₀	0.34	1.78	2.42
S₁	0.70	2.93	3.81
LSD_{5%}	*	0.25	0.32
note: * not statistically analysed			

As can be seen in Table 3.3, S fertilisation significantly increased ($p < 0.001$) the $\text{SO}_4\text{-S}$ content in the leaf tissue of winter oilseed rape. A mean increase by 3.9-fold in Braunschweig, by 1.8-fold in Aberdeen and by 3.5-fold in Inverness was determined (Table 3.3). The $\text{SO}_4\text{-S}$ content in young leaves showed also variation during the experimental seasons (Table 3.3). This indicates again different ranges of the S supply during the experimentation and/or different developmental stage of plants at the sampling date.

The analysis of variance revealed no significant effect of S applications on the total N content in young leaves of winter oilseed rape at the start of stem elongation. These results are not shown in this chapter (see Appendix: Table A.9 to A.11).

Dependence of mineral composition and seed yield on the N supply

During experimentations, N fertilisation had little effect on the total S and $\text{SO}_4\text{-S}$ content in young fully developed leaves at the start of stem elongation, although there was a tendency for them to be slightly reduced by higher rates of N (see Appendix: Table A.9 to A.11). In the case of total S content in straw and pod walls, no significant influence of N fertilisation was found (see Appendix: Table A.12). On the other hand, high N applications significantly increased ($p < 0.05$) the total S content in seeds in almost all experimental seasons (exception Braunschweig 2001/2002 and Aberdeen 2001/2002) (Figure 3.1). This increase accounted for about 1.1-fold on all three experimental sites (mean over three seasons) (Figure 3.1). This may indicate that a higher supply of nitrate results in a higher uptake of sulphate by crops because of their mutual existence in proteins.

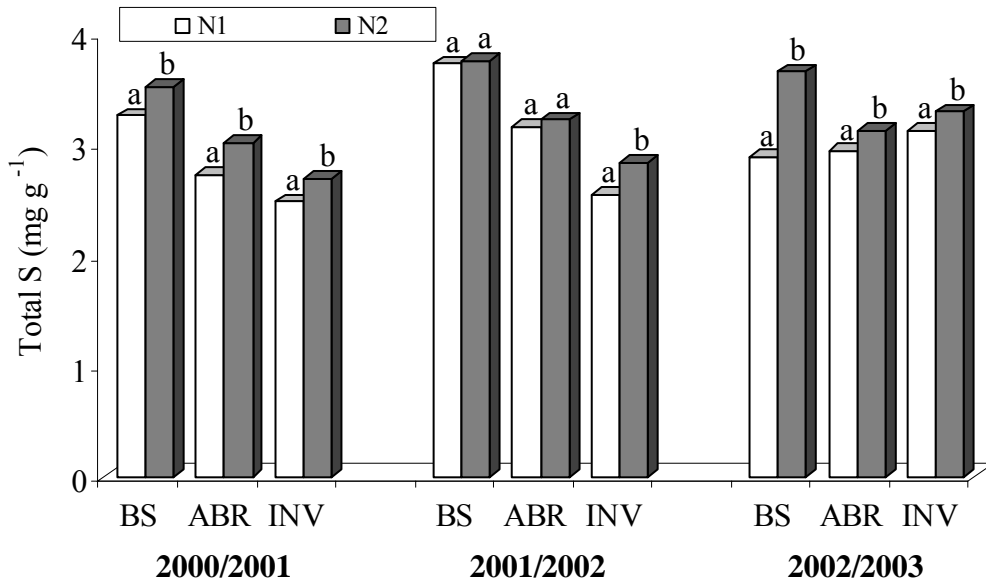


Figure 3.1: Effect of N fertilisation on the total S content in seeds of winter oilseed rape during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (different letters denote significant differences between N treatments at the 5% level by the t-test).

The effect of N fertilisation on the total N content in the leaf tissue of winter oilseed rape and on seed yield is presented in Table 3.4. As expected, the application of N significantly increased ($p < 0.001$) the total N content in young leaves and an average increase by about 1.2-fold was found on all three experimental sites (Table 3.4). Comparing the experimental seasons, variations were found with respect to leaves total N content (Table 3.4). This would seem to stem from different weather conditions, particularly different precipitation rates (see chapter 2.1 and Appendix: Table A.1 to A.3). The same pattern was observed in the case of total S and SO₄-S content since concomitant with sulphate nitrate is also leached (Schnug, 1997).

Regarding the seed yield, the application of N had a significant effect ($p < 0.01$) (exception: Braunschweig 2001/2002 and 2002/2003) (Table 3.4). The seed yield increased on average by 1.1-fold in Braunschweig and by 1.3-fold in Aberdeen and Inverness (Table 3.4).

Table 3.4: Effect of N fertilisation on the total N content in young fully developed leaves of winter oilseed rape at the start of stem elongation and on seed yield during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	Total N			Seed yield		
	mg g ⁻¹			t ha ⁻¹ (8% moisture)		
	BS	ABR	INV	BS	ABR	INV
2000/2001						
N ₁	50.5	51.1	50.3	3.33	3.17	2.34
N ₂	57.5	54.9	53.4	3.59	4.02	2.94
LSD _{5%}	1.74	1.03	*	0.21	0.12	0.18
2001/2002						
N ₁	40.6	44.7	45.9	2.05	2.86	2.15
N ₂	50.3	48.4	51.8	1.58	3.27	2.52
LSD _{5%}	1.10	1.88	1.94	0.27	0.21	0.16
2002/2003						
N ₁	35.8	36.3	37.8	2.92	3.04	2.43
N ₂	40.8	47.9	43.6	2.97	4.02	3.19
LSD _{5%}	1.58	1.14	1.88	0.11	0.16	0.38
note: * not statistically analysed						

Varietal differences and effect of fungicide treatment on mineral composition and seed yield

The analysis of variance showed no significant differences between cultivars with respect to the mineral content and also no significant effect of the fungicide treatment on mineral composition (see Appendix: Table A.9 to A.12).

Regarding the seed yield, the cultivar *Lipton* yielded more than the cultivar *Bristol* (exception Inverness 2000/2001), but differences were not all the time significant (Figure 3.2). The average yield for the cultivar *Lipton* was 2.9 t ha⁻¹, 3.6 t ha⁻¹ and 2.7 t ha⁻¹ in Braunschweig, Aberdeen and Inverness, respectively, whereas for the cultivar *Bristol* the corresponding values were 2.6 t ha⁻¹ in Braunschweig, 3.2 t ha⁻¹ in Aberdeen and 2.5 t ha⁻¹ in Inverness (Figure 3.2). When one considers that during the experimentation *P. brassicae* was the most important disease, the lower yields found for *Bristol* support the predicted susceptibility of this cultivar to *P. brassicae*.

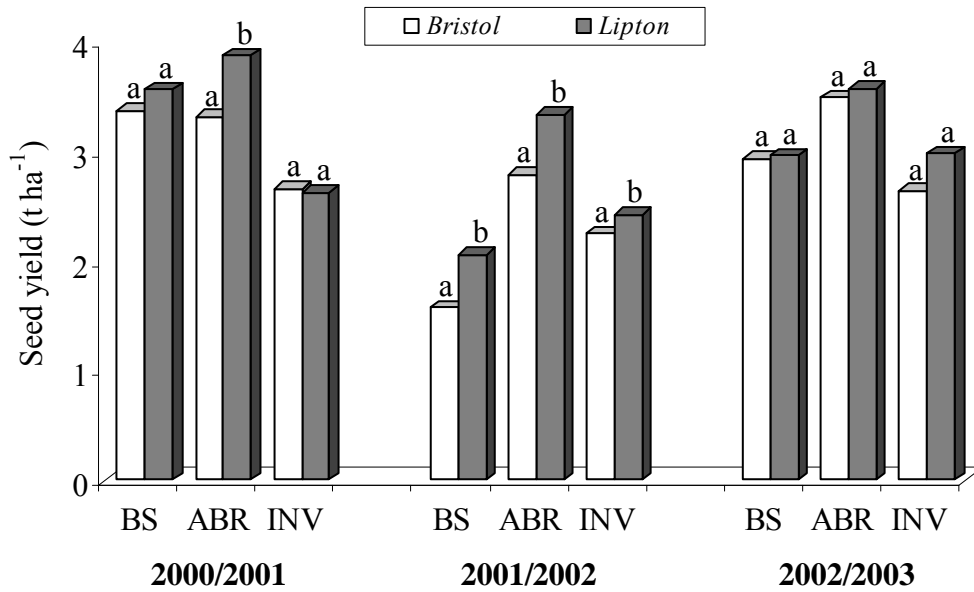


Figure 3.2: Effect of cultivar on seed yield of winter oilseed rape during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (different letters denote significant differences between cultivars at the 5% level by the t-test).

The yield was also increased by the fungicide treatment, but again differences were not statistically significant in all experimental seasons (Figure 3.3). The average yield response to fungicide applications was 13.4% in Braunschweig, 4.8% in Aberdeen and 14.7% in Inverness (Figure 3.3).

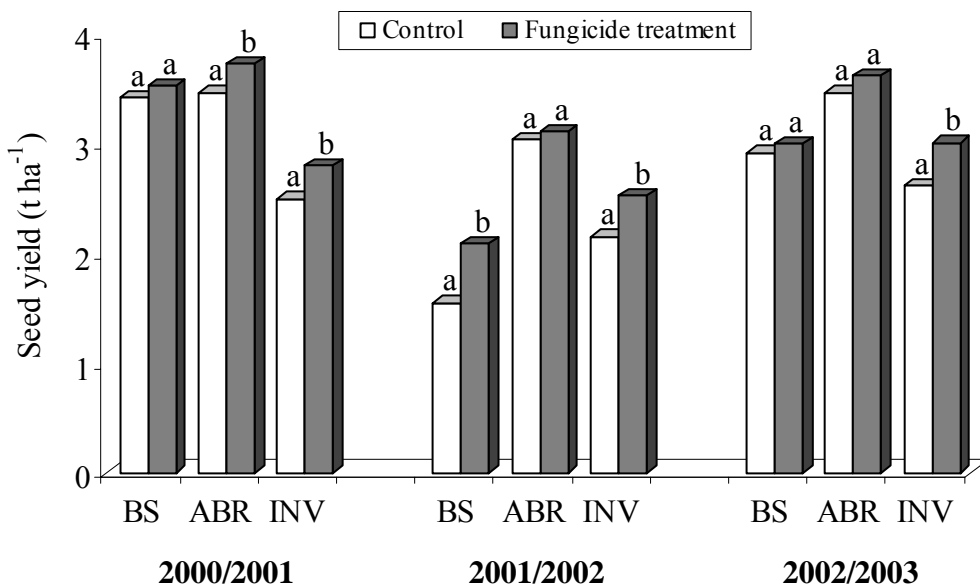


Figure 3.3: Effect of fungicide treatment on seed yield of winter oilseed rape during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (different letters denote significant differences between fungicide treatments at the 5% level by the t-test).

Conversion factors for the effect of sulphur and cultivar on mineral S fractions and seed yield

As outlined above, differences were observed between sites and seasons with respect to the investigated mineral S fractions and yield. Since S was applied at different timings and doses during the experimentation, comparisons between sites and seasons are hardly to be made by simply testing the absolute increase/decrease of the plant parameters as a result of S fertilisation. The application of the “principle of equal treatment of all test subjects” is used as the basis of many comparative tests (Finck, 1982). Conversion factors have been therefore calculated, which show the increase/decrease of the investigated parameter per each kilogram of S applied to the soil. Conversion factors for the effect of S and cultivar on mineral S fractions in leaves and seeds and on seed yield are summarised in Table 3.5.

Table 3.5: Conversion factors for the effect of S on mineral S fractions and seed yield of two winter oilseed rape cultivars during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Variable	<i>Bristol</i>			<i>Lipton</i>		
	<i>BS</i>	<i>ABR</i>	<i>INV</i>	<i>BS</i>	<i>ABR</i>	<i>INV</i>
2000/2001						
Leaf S (mg/g)	0.049	0.029	0.032	0.034	0.027	0.028
Leaf SO₄-S (mg/g)	0.037	0.036	0.033	0.026	0.020	0.021
Seed S (mg/g)	0.002	0.004	0.007	0.003	0.001	0.005
Seed yield (t/ha)	0.002	-0.0002	0.006	0.002	0.0007	0.0003
2001/2002						
Leaf S (mg/g)	0.029	0.038	0.042	0.031	0.031	0.042
Leaf SO₄-S (mg/g)	0.009	0.005	0.011	0.010	0.006	0.010
Seed S (mg/g)	0.006	0.004	0.002	0.004	0.003	0.004
Seed yield (t/ha)	0.0006	0.002	-0.0002	0.002	0.002	0.002
2002/2003						
Leaf S (mg/g)	0.018	0.014	0.011	0.016	0.010	0.008
Leaf SO₄-S (mg/g)	0.003	0.012	0.017	0.002	0.011	0.011
Seed S (mg/g)	0.006	0.003	0.004	0.005	0.002	0.003
Seed yield (t/ha)	0.0004	-0.0008	0.001	-0.0006	0.0008	0.00004

As can be seen in Table 3.5, in the third season S fertilisation had a small effect on mineral S content and seed yield (Table 3.5). In the first and second season, the total S content in young leaves increased on average by 0.033 mg S g⁻¹ and 0.036 mg S g⁻¹, respectively per each kg of S applied (mean over three sites) (Table 3.5). On the other hand, in the third season, an average increase of just 0.013 mg S g⁻¹ was noted (mean over three sites) (Table 3.5). The same pattern was found for the SO₄-S content in young leaves and the seed yield, whereas in the case of total S content in seeds differences between seasons were not as strong (Table 3.5). The cause for the seasonal variability of the S supply might be due to several factors such as deficiency of other nutrients (see Appendix: Table A.9 to A.11), weather conditions (see chapter 2.1 and Appendix: Table A.1 to A.3), the developmental stage of plants at the sampling date, infections of oilseed rape crop by fungal pathogens (see chapter 3.3, 3.4 and 3.5).

Comparing the experimental sites, it can be stated that S had the greatest effect on the very low S site in Inverness compared to Aberdeen (Table 3.5). For instance, the total S content in young leaves increased on average by 0.03 mg S g⁻¹ in Inverness and by 0.02 mg S g⁻¹ in Aberdeen per each kg of S applied. Compared to Braunschweig no general statements can be made.

Small differences between cultivars were noted with respect to the S acquisition (Table 3.5). Whereas for the cultivar *Bristol* the total S content in leaves increased on average by 0.029 mg S g⁻¹ per each kg of S applied, for the cultivar *Lipton* the corresponding value is 0.025 mg S g⁻¹ (mean over three sites and three seasons) (Table 3.5).

The results of the GLM procedure revealed that S fertilisation did not significantly increase the seed yield, while N fertilisation and fungicide treatment had a significant effect on crop productivity. Differences between cultivars were found with respect to the final seed yield, with higher yields determined for the cultivar *Lipton*.

Mineral concentrations in winter oilseed rape plants presented large variations depending on sites and seasonal weather conditions. However, there was a significant compositional effect of S applications on the total S content in leaves, straw, pod walls and seeds. Generally, the very low S site in Inverness showed the highest responses to S applications with respect to all investigated parameters. High rates of N fertilisation increased the total N content in young leaves and the total S content in seeds.

3.2 Influence of sulphur and nitrogen fertilisation, cultivar and fungicide treatment on sulphur-containing primary and secondary compounds of winter oilseed rape

As outlined in chapter 1.2, the S nutrition of plants interacts with their resistance against pests and diseases by controlling the plant vigour and consequently enhancing the concentration of S metabolites, which are supposed to be relevant for *SIR* (De Kok et al., 1981; Schnug and Ceynowa, 1990; Schnug et al., 1995b, Wielebski et al., 1999). The objective of this part of the research work is to quantify the extent to which S and N fertilisation, cultivar and fungicide treatment affect the content of primary and secondary S compounds in young leaves and seeds of winter oilseed rape at three different locations. Among organic S fractions, attention has been focussed on cysteine, GSH, GSLs and protein-S. Individual data for the S-containing primary and secondary compounds are listed in the appendix (Table A.13 to A.15).

Since the fungicide treatment delivered no statistical differences with respect to the cysteine, GSH, GSL and protein-S content, its effect is not shown in this chapter (see Appendix: Table A.13 to A.15).

Dependence of organic S fractions on the S supply

Free cysteine and GSH are S-containing compounds of the primary metabolism. These metabolites are supposed to be involved in plant resistance mechanisms against fungal pathogens (Vidhyasekaran, 2000; Gullner and Kömives, 2001). Previous studies also revealed a close relationship between plant S status and the cysteine and GSH content (Bosma et al., 1990; De Kok, 1990; Schnug et al., 1995b; Schnug 1997).

The effect of S fertilisation on the cysteine and GSH content in young fully developed leaves of winter oilseed rape at the start of stem elongation during three years research period is shown in Table 3.6. The application of S significantly increased ($p < 0.001$) the free cysteine content in the leaf tissue (Table 3.6). The mean increase accounted for 1.9-fold in Braunschweig and Inverness, whereas in Aberdeen an increase by 1.3-fold was found. On the other hand, the effect of S fertilisation on the GSH content was not regular: in some years the GSH content was higher in S fertilised plots, while in others a lower content was found in plants from these plots (Table 3.6). It has been also become obvious that the GSH content had a very large variability (see Appendix: Table A.13 to A.15). This indicates that the GSH concentration was not only dependent on the S supply, but also on a wide number of environmental factors (e.g. fungal infections; see chapter 3.4 and 3.5). The mean GSH content in young fully developed leaves at the start of stem elongation ranged from $12.1 \mu\text{mol g}^{-1}$ to $23.4 \mu\text{mol g}^{-1}$ in Braunschweig, from $11.0 \mu\text{mol g}^{-1}$ to $27.5 \mu\text{mol g}^{-1}$ in Aberdeen, and from $10.6 \mu\text{mol g}^{-1}$ to $30.5 \mu\text{mol g}^{-1}$ in Inverness

(Table 3.6). These values are higher by about 25-fold than the GSH concentrations reported for oilseed rape in greenhouse experiments of Schnug et al. (1995b) or the ranges found in other plant species (De Kok et al., 1981).

Comparing the experimental seasons, differences were noted with respect to the cysteine and GSH content (Table 3.6). This can be traced back to different availabilities of S to plants due to different seasonal climatic conditions (see chapter 2.1 and Appendix: Table A.1 to A.3) or to different infection rates of fungal pathogens. For instance, in the second season in Braunschweig, infections of oilseed rape by *P. brassicae* and *L. maculans* were observed (see Table 2.5). This might explain the higher cysteine and GSH concentration found in the leaf tissue of winter oilseed rape in this season (Table 3.6). The influence of fungal infections on the cysteine and GSH pool is presented in the next chapters (see chapter 3.4 and 3.5).

Table 3.6: Effect of S fertilisation on the cysteine and glutathione (GSH) content in young fully developed leaves of winter oilseed rape at the start of stem elongation during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	Cysteine			Glutathione		
	$\mu\text{mol g}^{-1}$					
	BS	ABR	INV	BS	ABR	INV
2000/2001						
S₀	0.81	1.04	0.81	12.1	19.5	11.9
S₁	2.10	1.23	1.46	15.2	19.2	11.9
LSD_{5%}	0.17	*	0.11	2.40	*	1.71
2001/2002						
S₀	1.42	0.62	0.50	23.4	21.1	12.9
S₁	2.57	0.91	1.25	20.4	27.5	30.5
LSD_{5%}	0.17	0.09	0.16	5.49	1.39	5.03
2002/2003						
S₀	0.65	0.71	0.54	14.8	11.3	14.2
S₁	0.90	0.83	0.74	15.2	11.0	10.6
LSD_{5%}	*	0.05	0.12	*	1.69	1.87
note: * not statistically analysed						

Glucosinolates are S-containing secondary compounds. The importance of the S supply for the GSL content has already been stressed by Schnug (1988). It is also assumed that these metabolites are an important part of the constitutive defence mechanisms of GSL-containing plants against fungal pathogens (Schnug and Ceynowa, 1990; Doughty et al., 1991).

The effect of S fertilisation on the total GSL content in young fully developed leaves and seeds of winter oilseed rape during three years research period is summarised in Table 3.7.

Table 3.7: Effect of S fertilisation on the total glucosinolate (GSL) content in young fully developed leaves and seeds of winter oilseed rape during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	<i>Total GSL</i>					
	$\mu\text{mol g}^{-1}$					
	<i>Leaves</i>			<i>Seeds</i>		
	<i>BS</i>	<i>ABR</i>	<i>INV</i>	<i>BS</i>	<i>ABR</i>	<i>INV</i>
2000/2001						
S₀	3.21	3.78	5.88	7.18	5.75	2.44
S₁	4.56	4.05	7.90	10.4	7.76	6.57
LSD_{5%}	0.49	*	0.77	0.46	0.32	0.56
2001/2002						
S₀	5.45	3.83	2.59	10.2	5.97	3.41
S₁	5.92	4.35	4.20	15.3	8.21	9.36
LSD_{5%}	1.31	0.95	0.73	0.86	0.67	0.45
2002/2003						
S₀	1.49	4.26	2.49	4.78	8.76	7.69
S₁	2.25	4.97	3.11	10.4	11.1	10.2
LSD_{5%}	*	0.49	0.65	0.87	0.79	0.57
note: * not statistically analysed						

The concentration of GSLs was much higher in seeds compared to the vegetative material (exception Inverness 2000/2001) (Table 3.7). In general, S applications significantly increased the total GSL content in leaves and seeds of winter oilseed rape (Table 3.7). It appears therefore feasible to enhance the natural resistance of oilseed rape plants against pests and diseases by S fertilisation. The average increase of the total GSL content in leaves was 1.3-fold in

Braunschweig, 1.1-fold in Aberdeen, and 1.4-fold in Inverness. For seeds, the corresponding values were 1.7-fold, 1.3-fold and 2.3-fold in Braunschweig, Aberdeen and Inverness, respectively.

During experimentations, the mean total GSL content in the leaf tissue ranged from 1.5 $\mu\text{mol g}^{-1}$ to 7.9 $\mu\text{mol g}^{-1}$, whereas in seeds varied between 2.4 $\mu\text{mol g}^{-1}$ and 15.3 $\mu\text{mol g}^{-1}$. Compared to the field experiments conducted by Booth et al. (1991) in Scotland, the GSL concentrations in leaves and seeds are rather small.

As can be seen in Table 3.7, there were marked differences between sites and seasons with respect to the total GSL content. The GSL content in rapeseed from control plots was higher in Braunschweig (7.4 $\mu\text{mol g}^{-1}$) compared to Aberdeen and Inverness (6.8 $\mu\text{mol g}^{-1}$ and 4.5 $\mu\text{mol g}^{-1}$, respectively) (Table 3.7). This again follows the ranking of the sites according to their potential to supply S to the plants.

S fertilisation had a positive impact on the content of individual GSLs as well. Earlier studies have shown that different GSLs exhibit different activity on fungal pathogens (Peterka and Schloesser 1989; Schnug and Ceynowa 1990). Moreover, alkenyl GSLs are supposed to participate in the general resistance of plants against fungal diseases, whereas indole and aromatic GSLs in the active (“induce”) protection (Zukalová and Vašák, 2002).

In leaves and seeds of winter oilseed rape three predominant alkenyl GSLs were detected: glucobrassicinapin (4-pentenyl glucosinolate), gluconapin (3-butenyl glucosinolate) and progoitrin (2-hydroxy-3-butenyl glucosinolate). The application of S tended to be associated with an increase of this type of GSLs, findings that are in accordance with previous investigations (Schnug, 1988; Zhao et al., 1994). The results from the second year of experimentation are shown in Figure 3.4 and 3.5. The predominant alkenyl GSL in young leaves of winter oilseed rape at the start of stem elongation was glucobrassicinapin, followed by progoitrin and gluconapin (Figure 3.4). Glucobrassicinapin was reported to have biocidal properties (Peterka and Schloesser, 1989) and thus, the high concentration of this GSL in the leaf tissue might have some protective function against fungal pathogens. The average increase of the alkenyl GSL contents in leaves as a result of S fertilisation was 1.3-fold for glucobrassicinapin, and gluconapin, and 1.4-fold for progoitrin (mean over three sites) (Figure 3.4).

In the case of seeds, the highest fraction of alkenyl GSLs was represented by progoitrin, followed by gluconapin and glucobrassicinapin (Figure 3.5). The progoitrin content in seeds increased in the second season on average by 1.9-fold, gluconapin by 2.0-fold and glucobrassicinapin by 2.1-fold when S was applied to the soil (mean over three sites) (Figure 3.5).

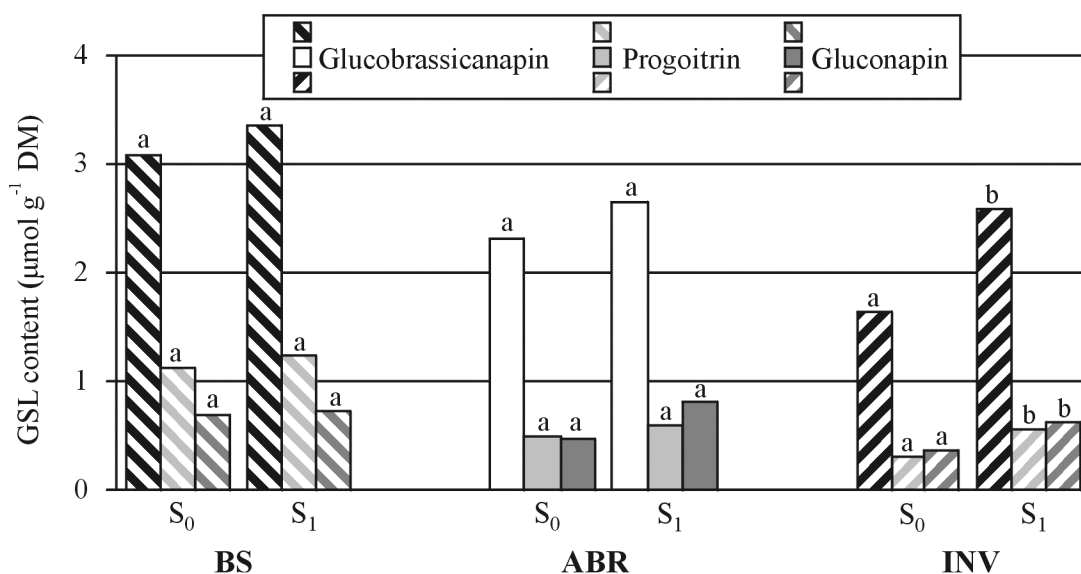


Figure 3.4: Main alkenyl glucosinolate (GSL) content in young fully developed leaves of winter oilseed rape at the start of stem elongation in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between S treatments at the 5% level by the t-test).

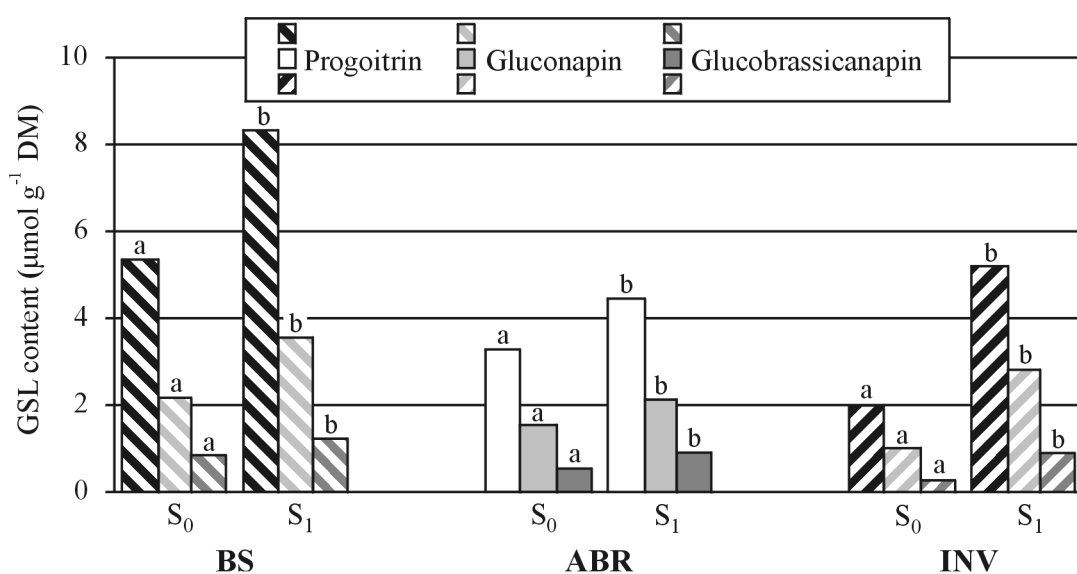


Figure 3.5: Main alkenyl glucosinolate (GSL) content in seeds of winter oilseed rape in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between S treatments at the 5% level by the t-test).

The indole and aromatic GSLs made a larger contribution to the total GSL content in the vegetative tissues than in seeds. Glucobrassicin (3-indole methyl glucosinolate) and gluconasturtiin (2-phenyl ethyl glucosinolate) were the main indole and aromatic GSLs in the leaf tissue. On the other hand, 4-hydroxy glucobrassicin (4-hydroxy-3-indole methyl glucosinolate) accounted for the largest fraction of the indole GSLs in rapeseed. The role of indole and aromatic GSLs from seeds seems to be of lesser interest in the discussion about *SIR* of plants against fungal pathogens. Therefore, in this chapter only the effect of S fertilisation on the glucobrassicin and gluconasturtiin content in young fully developed leaves of winter oilseed rape at the start of stem elongation is shown (Figure 3.6). The effect of S fertilisation on the indole and aromatic GSL content in seeds is shown in the appendix (Table A.13 and A.15). As can be seen in Figure 3.6, S fertilisation increased the glucobrassicin and gluconasturtiin content in the leaf tissue. The average increase was 1.6-fold for glucobrassicin and 1.4-fold for gluconasturtiin (mean over three sites) (Figure 3.6).

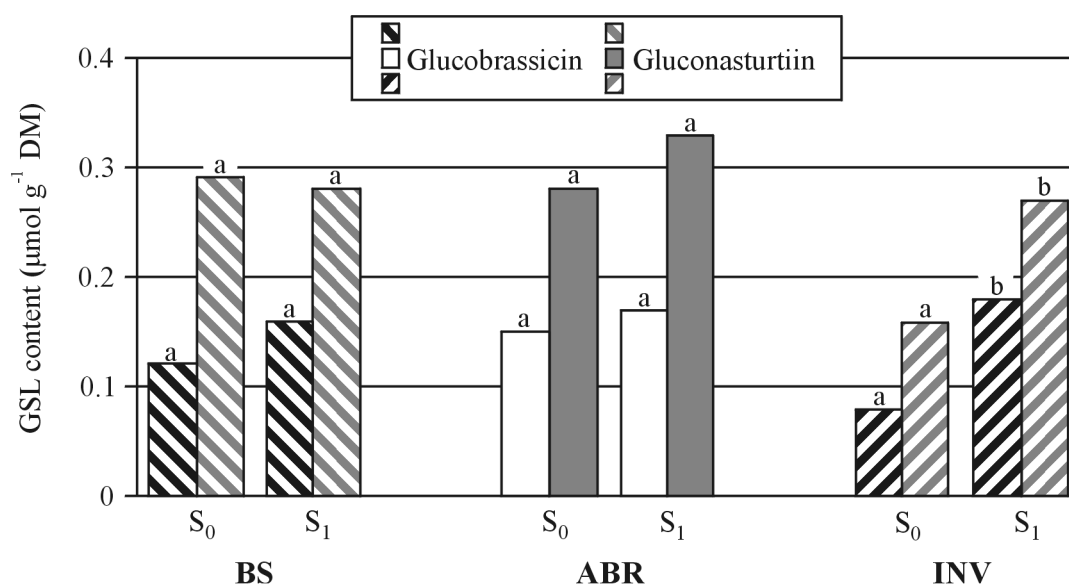


Figure 3.6: Main indole and aromatic glucosinolate (GSL) content in young fully developed leaves of winter oilseed rape at the start of stem elongation in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between S treatments at the 5% level by the t-test).

Other compounds considered to be an active part of the induced multiple plant defence mechanisms against microbial pathogens are pathogenesis-related proteins (PR proteins) (e.g. defensins and thionins) (van Loon et al., 1994). The total protein-S content was calculated as the

difference between total S content and S bound in SO_4^{2-} and organic S compounds (see chapter 2.4.2). The effect of S fertilisation on the protein-S content in young fully developed leaves of winter oilseed rape at the start of stem elongation is illustrated in Figure 3.7 (season 2001/2002). The analysis of variance revealed a significant effect of S fertilisation ($p < 0.05$) on the protein-S content in the leaf tissue (Figure 3.7). An average increase by 2.1-fold in Braunschweig, by 2.0-fold in Aberdeen and by 1.9-fold in Inverness was noted (Figure 3.7). However, when leaf samples were collected from plants at earlier developmental stages, the majority of the total S was found as SO_4^{2-} (e.g. Aberdeen 2000/2001; see Table 3.1 and 3.3). In this case, the protein-S represented a small part of the total S and no significant differences between control and S fertilised plots were noted (see Appendix: Table A.13 to A.15).

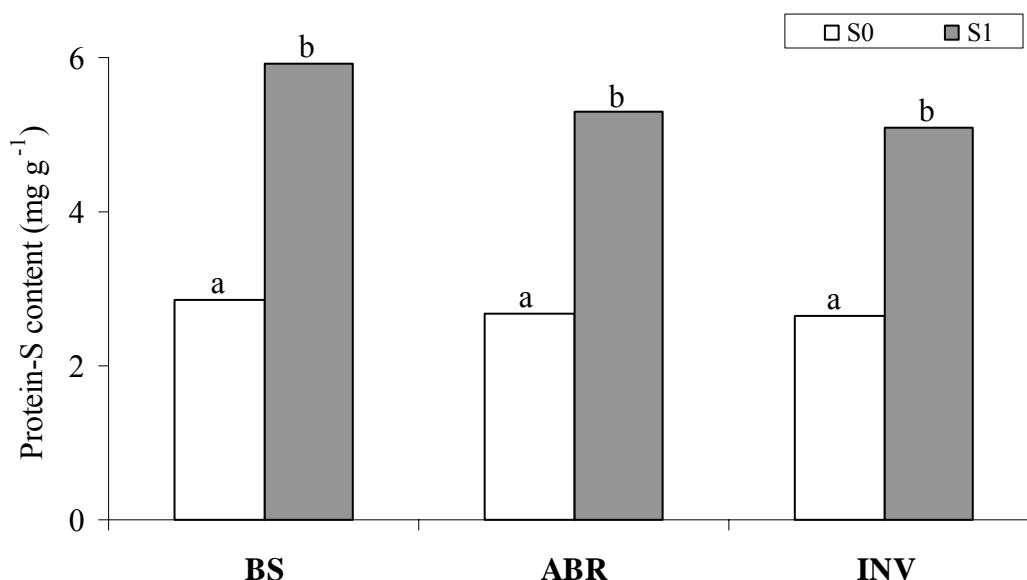


Figure 3.7: Effect of S fertilisation on the protein-S content in young fully developed leaves of winter oilseed rape at the start of stem elongation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between S treatments at the 5% level by the t-test).

Dependence of organic S fractions on the N supply

The effect of N fertilisation on the cysteine and GSH content in young leaves varied largely during the research period on all three sites (see Appendix: Table A.13 to A.15). Regarding the GSL contents, it has been observed that the application of high N rates tended to decrease the content of individual and total GSLs in young leaves (see Appendix: Table A.13 to A.15). In contrast, an increase of the GSL contents with higher N doses was found in seeds (see Appendix: Table A.13 to A.15). For the season 2001/2002, the effect of N fertilisation on the individual and

total GSL content in young leaves and seeds is illustrated in Figure 3.8 and 3.9, respectively. The application of 200 kg N ha⁻¹ as compared to 100 kg N ha⁻¹ depressed the total GSL content in leaves by about 22% (Figure 3.8) (mean over three sites), whereas the total GSL content in seeds increased by 6.0% (Figure 3.9) (mean over three sites).

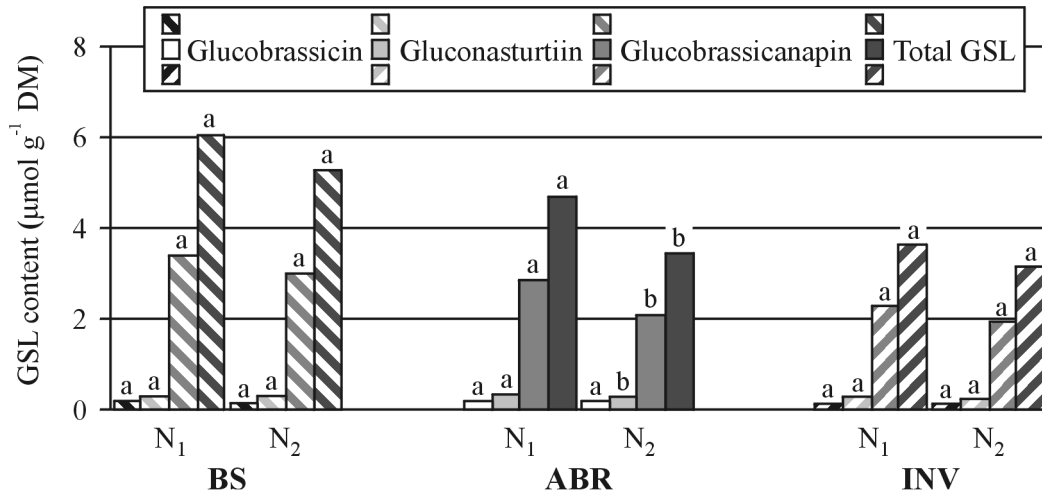


Figure 3.8: Effect of N fertilisation on the individual and total glucosinolate (GSL) content in young fully developed leaves of winter oilseed rape at the start of stem elongation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between N treatments at the 5% level by the t-test).

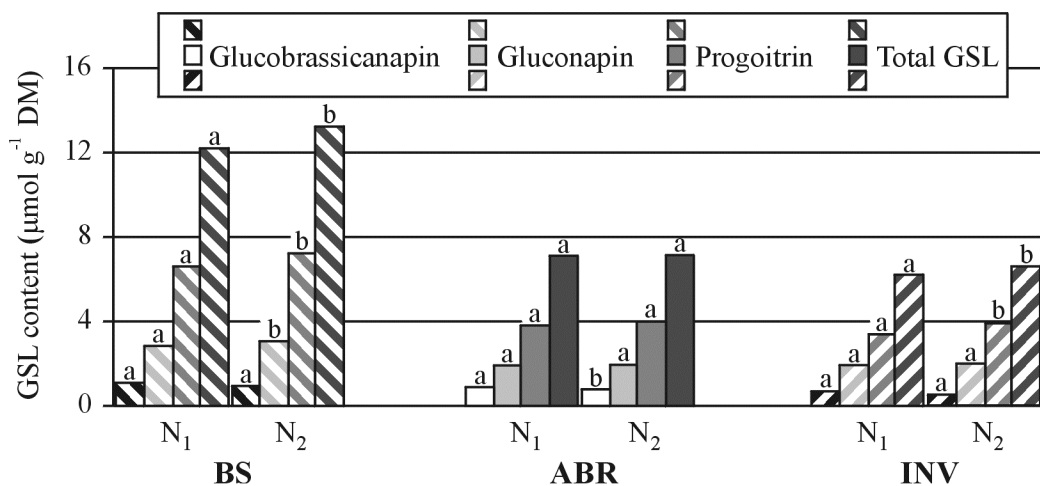


Figure 3.9: Effect of N fertilisation on the alkenyl and total glucosinolate (GSL) content in seeds of winter oilseed rape in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (different letters denote significant differences between N treatments at the 5% level by the t-test).

Differences in the content of organic S fractions between cultivars

Since cultivars selected for the present research work are reported to have different susceptibilities to fungal diseases, differences between cultivars with respect to the concentration of S-containing compounds putatively involved in stress resistance were investigated. The effect of cultivar on the cysteine and GSH content in young fully developed leaves of winter oilseed rape at the start of stem elongation is summarised in Table 3.8. The cultivar *Lipton* had a slightly lower cysteine content compared to the cultivar *Bristol* (exception Inverness 2001/2002) (Table 3.8). For *Lipton* a mean cysteine content of $1.0 \mu\text{mol g}^{-1}$ was found (mean over three sites and three seasons), whereas for *Bristol* the corresponding value was $1.1 \mu\text{mol g}^{-1}$. In the case of GSH content, no differences between cultivars could be noted (Table 3.8).

Table 3.8: Effect of cultivar on the cysteine and glutathione (GSH) content in young fully developed leaves of winter oilseed rape at the start of stem elongation during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	Cysteine			Glutathione		
	$\mu\text{mol g}^{-1}$					
	BS	ABR	INV	BS	ABR	INV
2000/2001						
<i>Bristol</i>	1.62	1.12	1.16	11.7	19.6	11.8
<i>Lipton</i>	1.29	1.10	1.11	15.7	19.1	12.0
LSD_{5%}	0.17	*	0.11	2.40	*	1.71
2001/2002						
<i>Bristol</i>	2.08	0.80	0.85	23.2	23.6	21.8
<i>Lipton</i>	1.90	0.73	0.90	20.5	25.0	21.6
LSD_{5%}	0.17	0.09	0.16	5.49	1.39	5.03
2002/2003						
<i>Bristol</i>	0.80	0.85	0.67	15.2	11.1	12.1
<i>Lipton</i>	0.76	0.70	0.61	14.8	11.2	12.6
LSD_{5%}	*	0.05	0.12	*	1.69	1.87
note: * not statistically analysed						

Among individual GSLs, differences between cultivars were found only with respect to the progoitrin content. The results from the statistical analysis are shown in Table 3.9. Generally, in both leaves and seeds, the cultivar *Lipton* contained a higher concentration of progoitrin than the cultivar *Bristol* (Table 3.9). The average progoitrin content in leaves was $0.6 \mu\text{mol g}^{-1}$ for *Bristol* and $0.8 \mu\text{mol g}^{-1}$ for *Lipton* (mean over three sites and three seasons), whereas in seeds the corresponding values were $4.0 \mu\text{mol g}^{-1}$ and $4.5 \mu\text{mol g}^{-1}$ for *Bristol* and *Lipton*, respectively.

Table 3.9: Effect of cultivar on the progoitrin content in young fully developed leaves and seeds of winter oilseed rape during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Treatment	<i>Progoitrin</i>					
	$\mu\text{mol g}^{-1}$					
	<i>Leaves</i>			<i>Seeds</i>		
	<i>BS</i>	<i>ABR</i>	<i>INV</i>	<i>BS</i>	<i>ABR</i>	<i>INV</i>
2000/2001						
<i>Bristol</i>	0.39	0.47	1.52	4.22	3.20	2.31
<i>Lipton</i>	0.82	0.67	1.57	5.23	3.50	2.63
LSD_{5%}	0.24	*	0.23	0.24	0.19	0.26
2001/2002						
<i>Bristol</i>	0.92	0.36	0.26	6.55	3.83	3.61
<i>Lipton</i>	1.38	0.71	0.55	7.18	3.95	3.57
LSD_{5%}	0.39	0.16	0.14	0.41	0.34	0.25
2002/2003						
<i>Bristol</i>	0.16	0.52	0.67	3.69	4.81	3.77
<i>Lipton</i>	0.61	0.48	0.72	6.02	4.42	4.18
LSD_{5%}	*	0.19	0.13	0.57	0.51	0.34
note: * not statistically analysed						

Conversion factors for the effect of sulphur and cultivar on organic S fractions

It has been outlined in chapter 3.1 that in order to compare sites and seasons the “relative S action” has to be determined. Therefore, conversion factors have been also calculated for the organic S fractions. As mentioned before, the conversion factor shows the increase/decrease of the investigated parameter per each kilogram of S applied to the soil. The results are summarised in Table 3.10.

Table 3.10: Conversion factors for the effect of S on organic S fractions in leaves (cysteine, GSH, GSL, protein-S) and seeds (GSL) of two winter oilseed rape cultivars during three years (2000-2003) in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV).

Variable	<i>Bristol</i>			<i>Lipton</i>		
	<i>BS</i>	<i>ABR</i>	<i>INV</i>	<i>BS</i>	<i>ABR</i>	<i>INV</i>
2000/2001						
Cysteine (µmol/g)	0.010	0.002	0.007	0.007	0.002	0.006
GSH (µmol/g)	-0.005	0.0004	-0.006	0.047	-0.005	0.005
GSL (µmol/g)	0.007	0.006	0.028	0.011	-0.004	0.012
Protein-S (mg/g)	0.011	-0.005	0.0008	0.005	0.008	-0.009
Seed GSL (µmol/g)	0.022	0.028	0.045	0.021	0.013	0.038
2001/2002						
Cysteine (µmol/g)	0.008	0.003	0.006	0.007	0.003	0.009
GSH (µmol/g)	-0.015	0.069	0.192	-0.026	0.061	0.161
GSL (µmol/g)	-0.002	0.005	0.013	0.008	0.005	0.019
Protein-S (mg/g)	0.019	0.029	0.018	0.021	0.023	0.025
Seed GSL (µmol/g)	0.042	0.027	0.067	0.027	0.018	0.052
2002/2003						
Cysteine (µmol/g)	0.002	0.001	0.002	0.002	0.0009	0.002
GSH (µmol/g)	0.004	0.004	-0.039	0.001	-0.0007	-0.033
GSL (µmol/g)	0.004	0.006	0.011	0.006	0.009	0.002
Protein-S (mg/g)	0.014	0.004	-0.002	0.013	-0.004	0.0003
Seed GSL (µmol/g)	0.040	0.031	0.032	0.035	0.015	0.018

The same pattern was found between experimental seasons as in the case of mineral S fractions: in the third season S fertilisation had a small effect. For instance, in the first and second season, the average increase of the cysteine content in young leaves of winter oilseed rape was $0.006 \mu\text{mol g}^{-1}$ per each kg of S applied (mean over three sites) (Table 3.10). For the total GSL content in the leaf tissue the corresponding values were $0.01 \mu\text{mol g}^{-1}$ for the first season and $0.008 \mu\text{mol g}^{-1}$ for the second season (Table 3.10). On the other hand, in the third season, the cysteine content increased by $0.002 \mu\text{mol g}^{-1}$ and the total GSL content by $0.006 \mu\text{mol g}^{-1}$ per each kg of S applied (mean over three sites) (Table 3.10). As outlined in chapter 3.1, the seasonal variability might be due to a wide number of factors that influence the plant growth and development under field conditions.

Comparing the experimental sites, it can be generally assumed that the greatest effect of S fertilisation on all investigated organic S fractions was at the Inverness site, where atmospheric S depositions are rated very low (Table 3.10). For example, in Inverness, the mean increase of the total GLS content in seeds was $0.04 \mu\text{mol g}^{-1}$ per each kg of S applied, whereas in Braunschweig and Aberdeen the corresponding figures were $0.03 \mu\text{mol g}^{-1}$ and $0.02 \mu\text{mol g}^{-1}$, respectively.

The cultivar *Bristol* reacted more sensitively to the S supply and therefore a slightly higher rise of the organic S fraction contents was found during the experimentation in comparison to the cultivar *Lipton* (Table 3.10). For instance, the GSL content in seeds increased on average by $0.04 \mu\text{mol g}^{-1}$ for *Bristol* and by $0.03 \mu\text{mol g}^{-1}$ for *Lipton* per each kg of S applied (mean over three sites and three seasons) (Table 3.10).

Correlation analysis

All S-containing primary and secondary compounds investigated in the present research work have a common precursor (i.e. cysteine). Thus, it was investigated if there are correlations between individual organic S fractions and between these fractions and mineral S contents. Differences between sites and experimental seasons were found. Detailed figures are not given for all individual experiments and two sites were chosen for comparison (individual data for all sites and seasons given in the appendix: Table A.9 to A.15).

In Figure 3.10 and 3.11, the relationships between leaf parameters for the experimental sites in Braunschweig (season 2001/2002) and Inverness (season 2001/2002) are shown. Highly significant positive relationships ($p < 0.01$) between the total S content and the cysteine ($r = 0.764$), GSH ($r = 0.616$), progoitrin ($r = 0.369$), glucobrassicinapin ($r = 0.442$), gluconapin ($r = 0.422$), glucobrassicin ($r = 0.504$), gluconasturtiin ($r = 0.372$), total GSL ($r = 0.468$) and $\text{SO}_4\text{-S}$ content ($r = 0.717$) were found in Inverness (Figure 3.11). On the other hand, in

Braunschweig, the total S content was highly significant correlated ($p < 0.001$) only with the cysteine ($r = 0.836$) and $\text{SO}_4\text{-S}$ content ($r = 0.592$) (Figure 3.10). Whereas in Inverness the cysteine content was strongly correlated with all S fractions (e.g. GSH, GSLs, $\text{SO}_4\text{-S}$) (Figure 3.11), in Braunschweig only correlations between the cysteine and $\text{SO}_4\text{-S}$ content were found ($r = 0.564$; $p < 0.001$) (Figure 3.10). Positive correlations between the GSH content with the individual and the total GSL content were observed in Inverness (Figure 3.11), but not in Braunschweig (Figure 3.10). Individual GSLs were highly significant correlated with each others and with the total GSL content at both sites (Figure 3.10 and 3.11). No significant or poor correlations were found between the individual or the total GSL content with the $\text{SO}_4\text{-S}$ content (Figure 3.10 and 3.11). The total N content was not correlated with the analysed S fractions (see Appendix: Table A.9 to A.15).

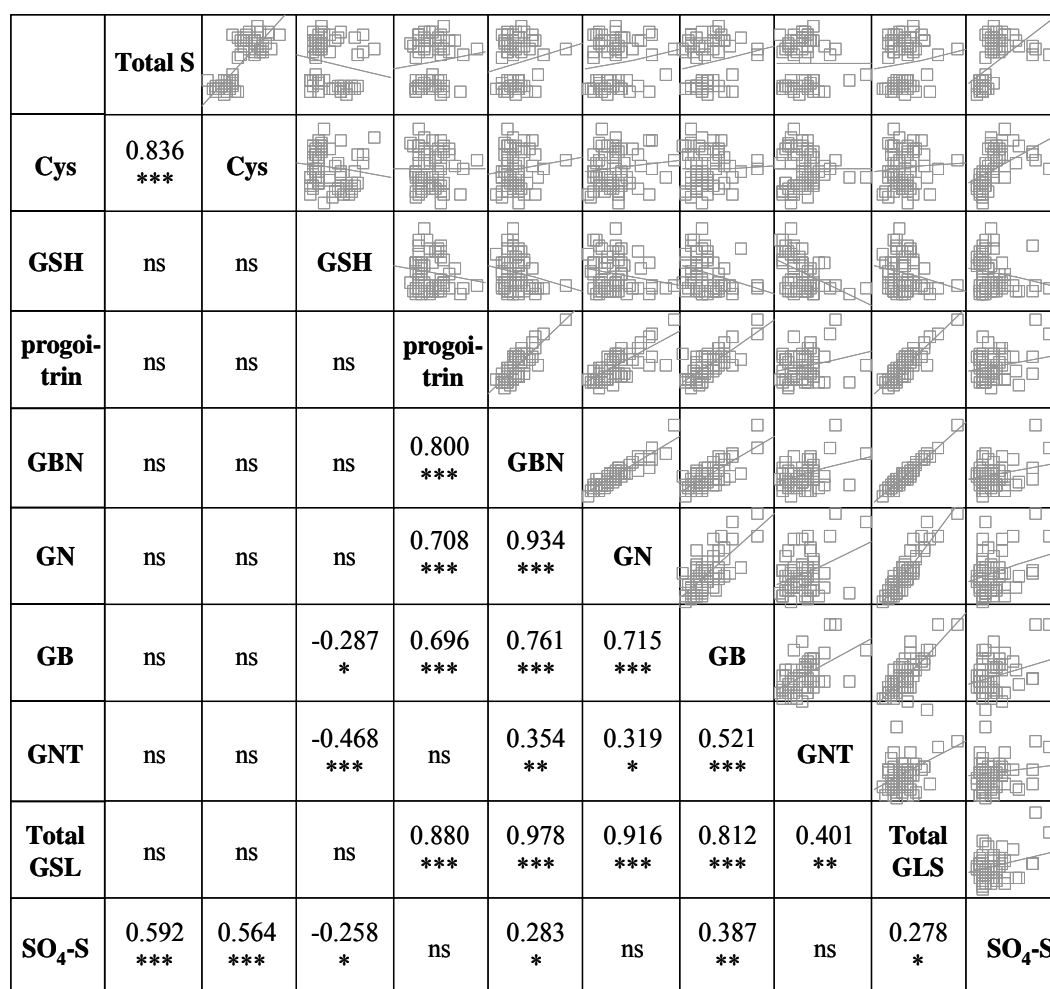


Figure 3.10: Correlation matrix for plant parameters in leaves of winter oilseed rape at the start of stem elongation in Braunschweig (season 2001/2002) (Cys – cysteine, GSH – glutathione; GBN – glucobrassicinapin; GN – gluconapin; GB – glucobrassicin; GNT – gluconasturtiin; Total GSL – total glucosinolates).

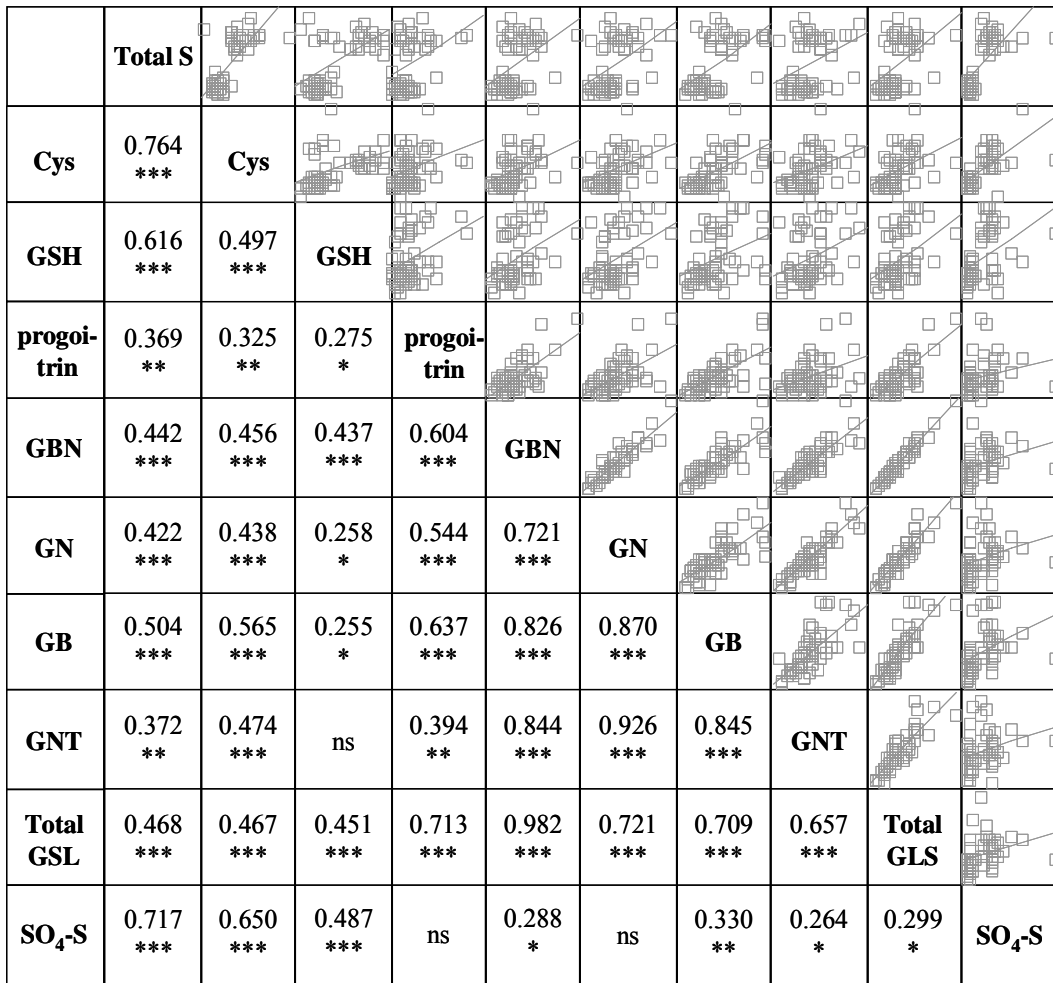


Figure 3.11: Correlation matrix for plant parameters in leaves of winter oilseed rape at the start of stem elongation in Inverness (season 2001/2002) (Cys – cysteine, GSH – glutathione; GBN – glucobrassicinapin; GN – gluconapin; GB – glucobrassicin; GNT – gluconasturtiin; Total GSL – total glucosinolates).

The relationships between seed parameters for the experimental sites in Braunschweig (season 2001/2002) and Inverness (season 2001/2002) are illustrated in Figure 3.12 and 3.13, respectively. Differences observed between experimental sites and seasons were not as strong as in the case of leaves. The total S content was positively correlated with the individual and the total GSL content at both sites (Figure 3.12 and 3.13). Strong or poor correlations were found between the individual and the total GSL content (Figure 3.12 and 3.13). In Braunschweig, highly significant correlations were found between the total S content in seeds and the total S content in straw ($r = 0.518$; $p < 0.001$) and pod walls ($r = 0.396$; $p < 0.001$), and between the total S content in straw and pod walls and the GSL content in seeds (Figure 3.12). Although in leaves no correlations were found between the total S and total N content (see Appendix: Table

A.9 to A.11), highly significant correlations ($p < 0.001$) were determined between these two parameters in straw ($r = 0.394$) and pod wall samples ($r = 0.589$) (Figure 3.12).

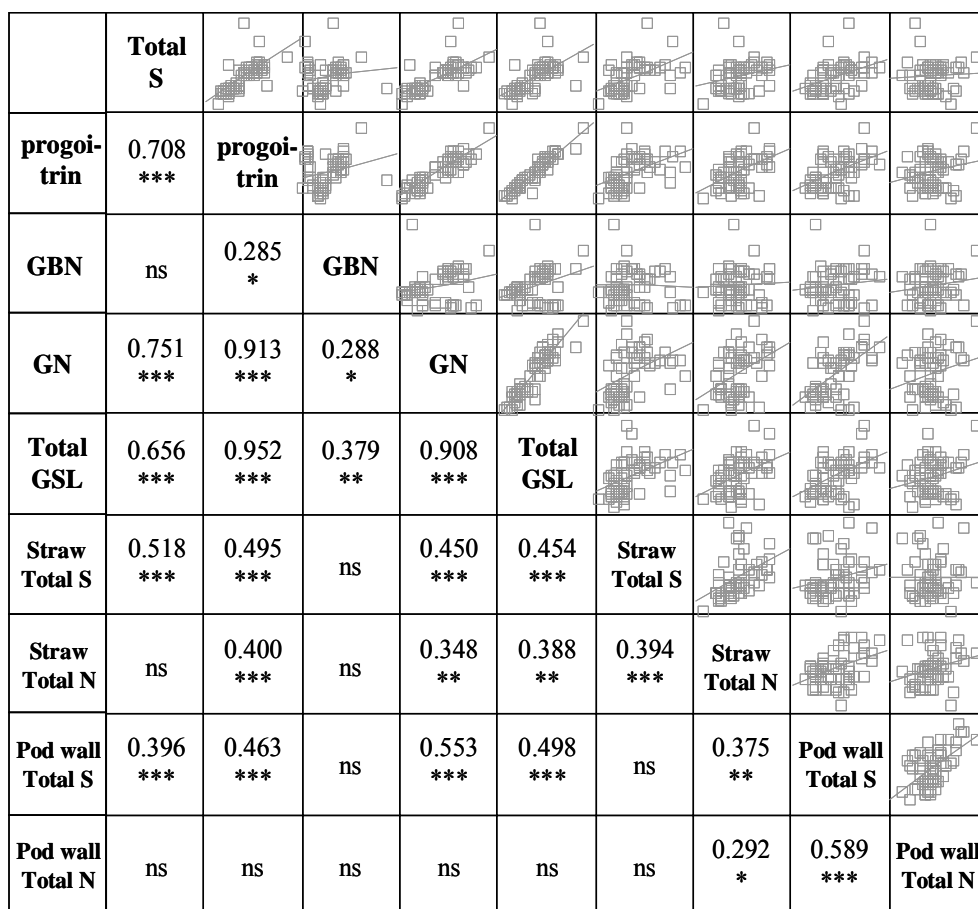


Figure 3.12: Correlation matrix for plant parameters in seeds, straw and pod walls of winter oilseed rape in Braunschweig (season 2001/2002) (GBN – glucobrassicinapin; GN – gluconapin; Total GSL – total glucosinolates).

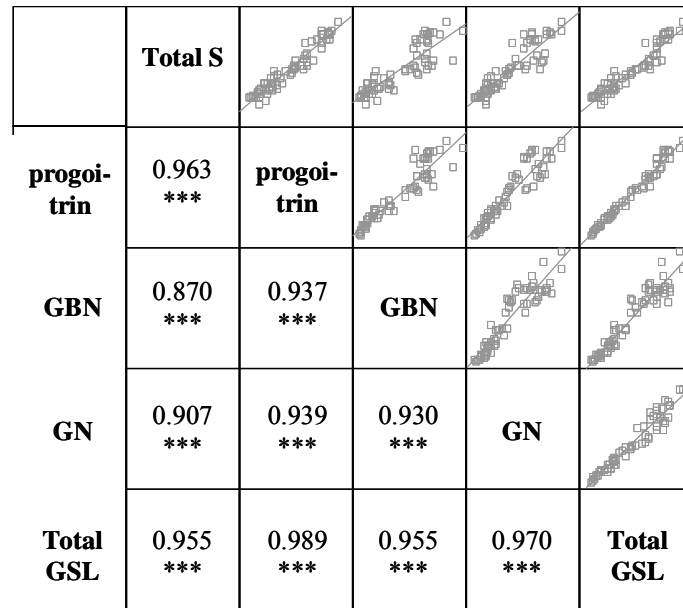


Figure 3.13: Correlation matrix for plant parameters in seeds of winter oilseed rape in Inverness (season 2001/2002) (GBN – glucobrassicinapin; GN – gluconapin; Total GSL – total glucosinolates).

Since S in plants is supposed to be continuously transported from one organ to another, the relationships between leaves, seeds, pod walls and straw parameters were also investigated. Correlations determined for the second season in Braunschweig are presented in Table 3.11. There were highly significant positive relationships ($p < 0.001$) between the total S content in leaves and the total S content in seeds ($r = 0.726$), straw ($r = 0.506$) and pod walls ($r = 0.546$) (Table 3.11). Additionally, the total S content in leaves was also highly significant correlated with the total GSL content in seeds at harvest ($r = 0.796$; $p < 0.001$) (Table 3.11). Significant correlations were also found between the $\text{SO}_4\text{-S}$ content in leaves with the total S and the total GSL content in seeds, and between the $\text{SO}_4\text{-S}$ content in leaves and the total S content in straw and pod walls (Table 3.11).

Table 3.11: Correlation coefficients for relationships between mineral S fractions in leaves and S fractions in seeds, straw and pod walls (Braunschweig, season 2001/2002).

	<i>Seed total S</i>	<i>Seed total GSL</i>	<i>Straw total S</i>	<i>Pod wall total S</i>
Leaf total S	$r = 0.726^{***}$	$r = 0.796^{***}$	$r = 0.506^{***}$	$r = 0.546^{***}$
Leaf $\text{SO}_4\text{-S}$	$r = 0.345^{**}$	$r = 0.413^{***}$	$r = 0.452^{***}$	$r = 0.266^*$

From the above results, it can be concluded that S fertilisation significantly increase the cysteine, GSL and protein-S content. A positive effect of S fertilisation on the GSH content was also found in some seasons. The content of organic S compounds showed variations depending on site and season. However, oilseed rape plants grown on the very low S site in Inverness generally showed the highest increases of the investigated S metabolites in response to S.

N fertilisation had no effect on the cysteine and GSH content. On the other hand, the N supply influenced the individual and the total GSL content: at high N levels a decrease of the GSL contents in leaves and an increase in seeds was found. However, modifications of the GSL levels in response to the N supply were not all the time significant.

Differences between cultivars were confined to the cysteine and progoitrin content, but not to GSH or other individual GSLs. The cultivar *Lipton* contained less cysteine and more progoitrin compared to the cultivar *Bristol*.

Significant correlations were found between all S fractions, but not in every season. Correlations were also found between leaf parameters with seed, pod wall and straw parameters.

3.3 Influence of sulphur applications on the level of fungal infections in winter oilseed rape crop

Work carried out in parallel at the Scottish Agricultural College (SAC), Aberdeen, Scotland aimed to investigate the potential of S fertilisation to reduce fungicide inputs for controlling *P. brassicae* in an oilseed rape field. During experimentations, on both sites in Scotland, oilseed rape plants showed symptoms of infection by various fungal pathogens such as *P. brassicae*, *L. maculans*, *P. parasitica*, *A. brassicae*, *B. cinerea*, *S. sclerotiorum*. The disease incidence and severity were scored at weekly or monthly intervals by the staff of the SAC and the data are summarised in the HGCA project no 326 (Sutherland et al., 2004).

Although previous investigations revealed that soil-applied S fertilisation significantly reduced the infection of crops by different fungal diseases (Schnug et al., 1995a; Bourbos et al., 2000; Klikocka et al., 2004), there are still open questions regarding the S doses require to induce S defence responses in plants and the timing of S applications. It is therefore the aim of this part of the research work to investigate the potential of two times S applications to inhibit or slow down fungal infections. The SAC working group involved in the project provided the disease assessment data displayed in this chapter. The disease assessment data before and after S fertilisation in spring, for the experimental seasons given in this chapter, are listed in the appendix (Table A.19 to A.21).

Pyrenopeziza brassicae was the most important disease in Scotland during the experimentation period. The development of *P. brassicae* throughout the growing season is illustrated in Figure 3.14. *Pyrenopeziza brassicae* appeared first in the mid-late November and increased to a maximum in the late March/April, at which time the disease incidence in non-fungicide treated plots was > 60% (Figure 3.14). Over three years research period, the incidence of *P. brassicae* (% plants infected at the start of stem elongation) was much higher at the low S status site in Aberdeen (91%) compared to the very low S status site in Inverness (51%) (Figure 3.14). The disease severity (% leaf area infected) followed the same pattern and the corresponding values were 14.6% and 5.8% in Aberdeen and Inverness, respectively (Figure 3.14). However, according to the HGCA founded project forecasting *P. brassicae* on winter oilseed rape (Steed and Fitt, 2000), any crop with greater than 25% of plants infected by *P. brassicae* at stem extension was deemed to have a severe infection. Thus, although at the Inverness site, where lower levels of *P. brassicae* infections were detected compared to the Aberdeen site, the epidemics were still severe. The extent of infection was similar for the resistant cultivar *Lipton* and the susceptible cultivar *Bristol* at the end of the experiment, and this contrast with their apparently different susceptibility to *P. brassicae* (Gladders et al., 1998; HGCA Recommended List WOSR 2003).

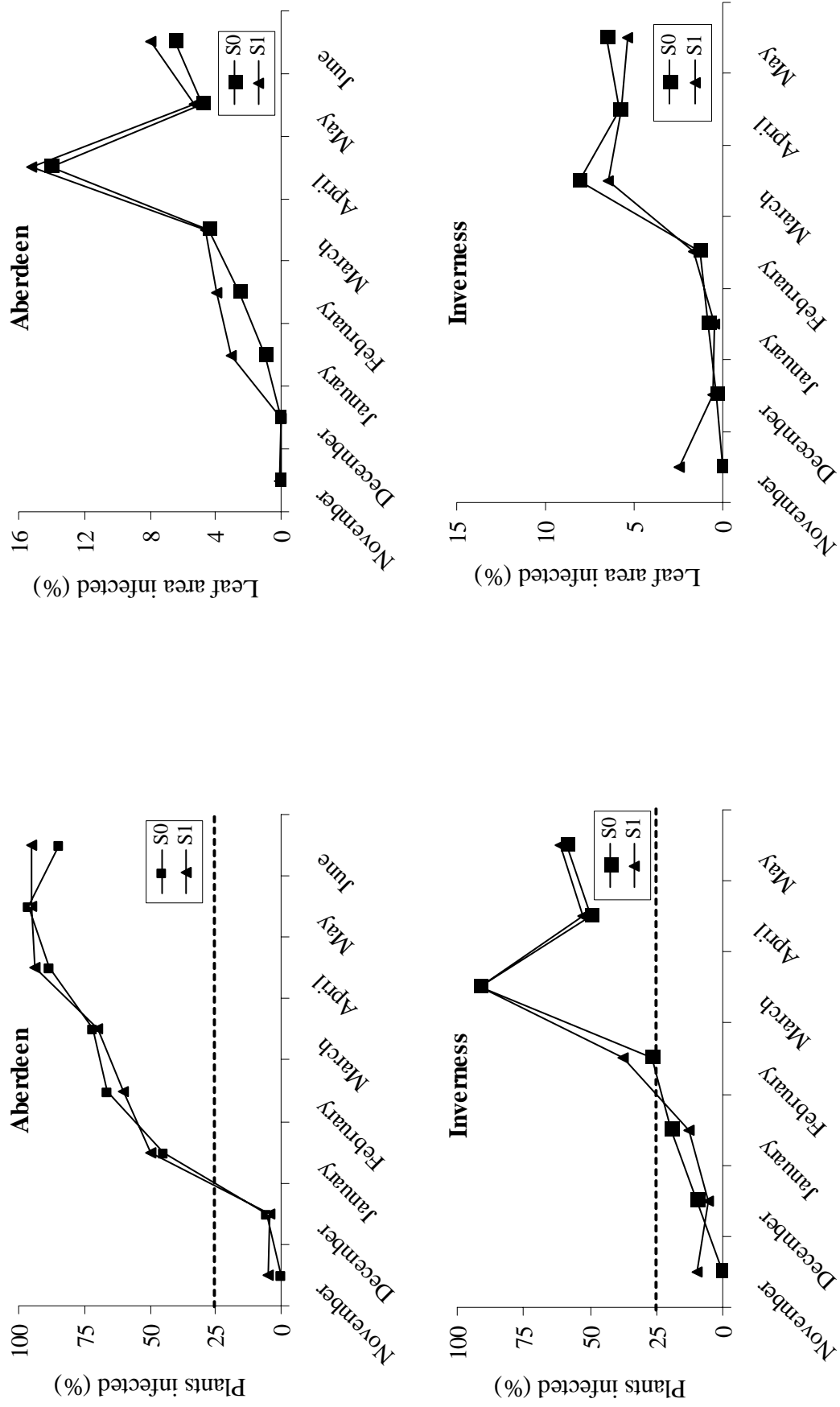


Figure 3.14: Development of *Pyrenopeziza brassicae* in winter oilseed rape crop (non-fungicide treated plots; mean over three seasons: 2000-2003).

Leptosphaeria maculans and *Peronospora parasitica* infections occurred in winter oilseed rape crop at low levels. The development of *L. maculans* and *P. parasitica* throughout the growing season is given in Figure 3.15 and 3.16, respectively. Slight *L. maculans* infections were observed in Inverness in the fall-winter period, while at the start of stem elongation no symptoms were detected. In comparison, in Aberdeen, *L. maculans* epidemics occurred during the whole growing season with values for disease incidence and severity of 2.9% and 0.01%, respectively (mean over three seasons at the start of stem elongation) (Figure 3.15). *Peronospora parasitica* infections occurred at slightly higher levels than *L. maculans* epidemics, with values for disease incidence of 4.6% and 2.9% in Aberdeen and Inverness, respectively (mean over three seasons at the start of stem elongation) (Figure 3.16). For disease severity, levels of 0.05% in Aberdeen and 0.06% in Inverness were found (mean over three seasons at the start of stem elongation) (Figure 3.16).

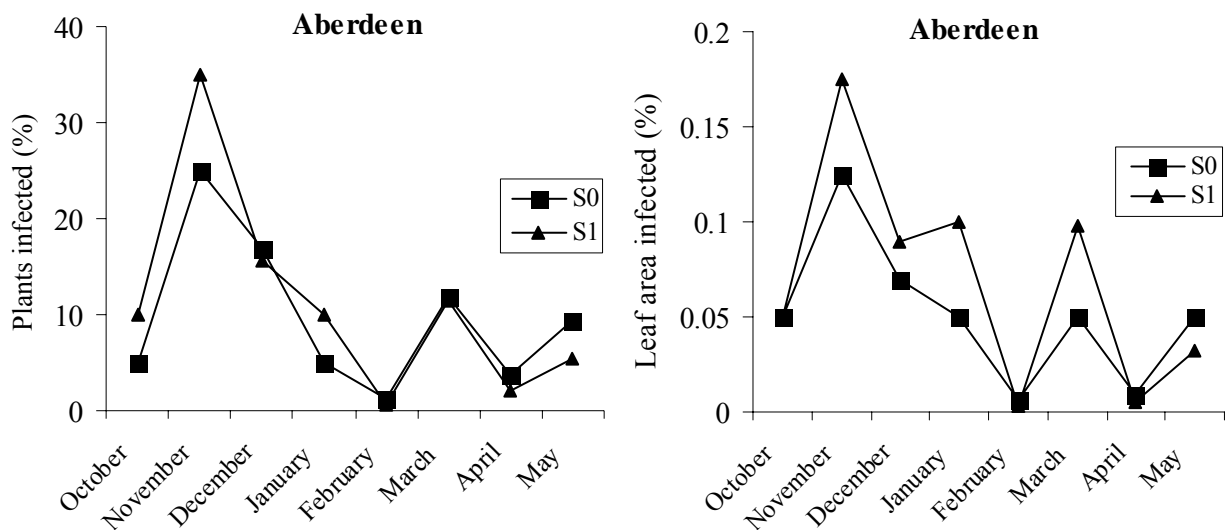


Figure 3.15: Development of *Leptosphaeria maculans* in winter oilseed rape crop (non-fungicide treated plots; mean over three seasons: 2000-2003).

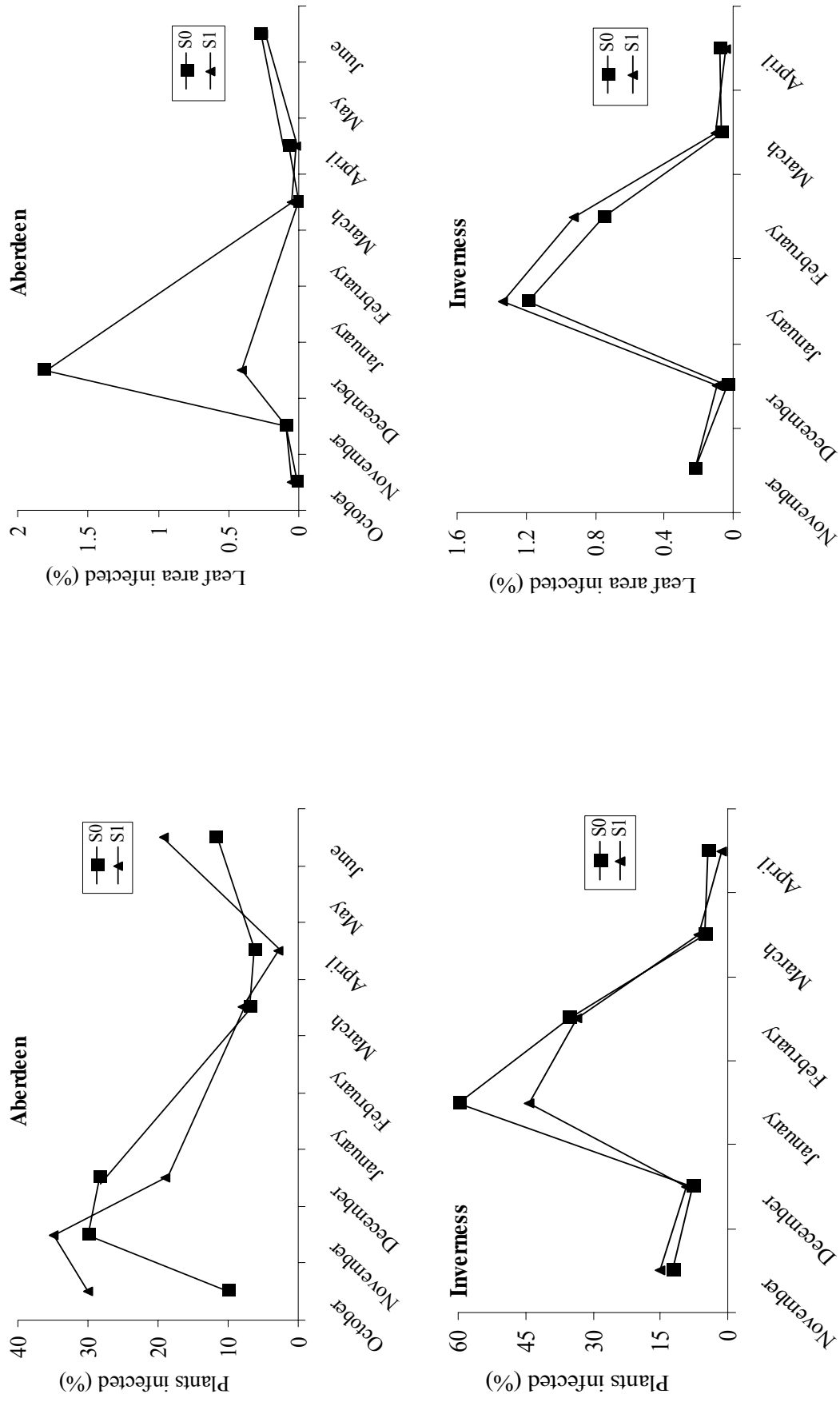


Figure 3.16: Development of *Peronospora parasitica* in winter oilseed rape crop (non-fungicide treated plots; mean over three seasons: 2000-2003).

The application of S in fall generally did not reliably delay the onset of *P. brassicae* development or reduce fall-winter infections in either variety at either site (data not shown). Also, no effect of fall S applications on *L. maculans* and *P. parasitica* infections was observed (data not shown).

The effect of spring S fertilisation on the disease incidence and severity of fungal pathogens at the Aberdeen and Inverness site is presented in Figure 3.17 and 3.18, respectively (season 2001/2002). The disease levels are given for two periods: before and after S fertilisation in spring. No significant differences were found between control and S fertilised plots and between cultivars with respect to the level of fungal infections at both sites (see Appendix: Table A.19 and A.20).

In the case of *P. brassicae* infections, spring S fertilisation had no effect on the disease incidence and severity of this pathogen (Figure 3.17 and 3.18). The infection level increased significantly from 44% to 94% of plants infected in Aberdeen (Figure 3.17) and from 85% to 100% of plants infected in Inverness (Figure 3.18). At both sites a significant increase in the disease severity of *P. brassicae* from 3% to 12% was also noted (Figure 3.17 and 3.18). In the case of infections caused by *L. maculans*, the disease incidence and severity was reduced in control as well as in S fertilised plots, but due to large inter-plot variations in disease levels a significant effect of S fertilisation on *L. maculans* could not be demonstrated (Figure 3.17 and 3.18). In contrast, the present investigations showed that the spring S application reduced the disease incidence and severity of *P. parasitica* with more than 50% at both sites (Figure 3.17 and 3.18). Therefore, when multiple pathogens were present on oilseed rape crop, spring S fertilisation tended to reduce infections caused by the biotrophic fungus *P. parasitica*, while infections caused by the hemi-biotrophic pathogen *P. brassicae* were enhanced.

Nevertheless, it might be possible that when single infections caused by hemi-biotrophs/necrotrophs are present on the crop, S applications may be successful. For instance, in Aberdeen in the third season, only infections of oilseed rape leaves by *P. brassicae* were observed at the time of spring S fertilisation. In this season, S tended to reduce the infection severity of this fungus, but no effect on the disease incidence was found (see Appendix: Table A.21).

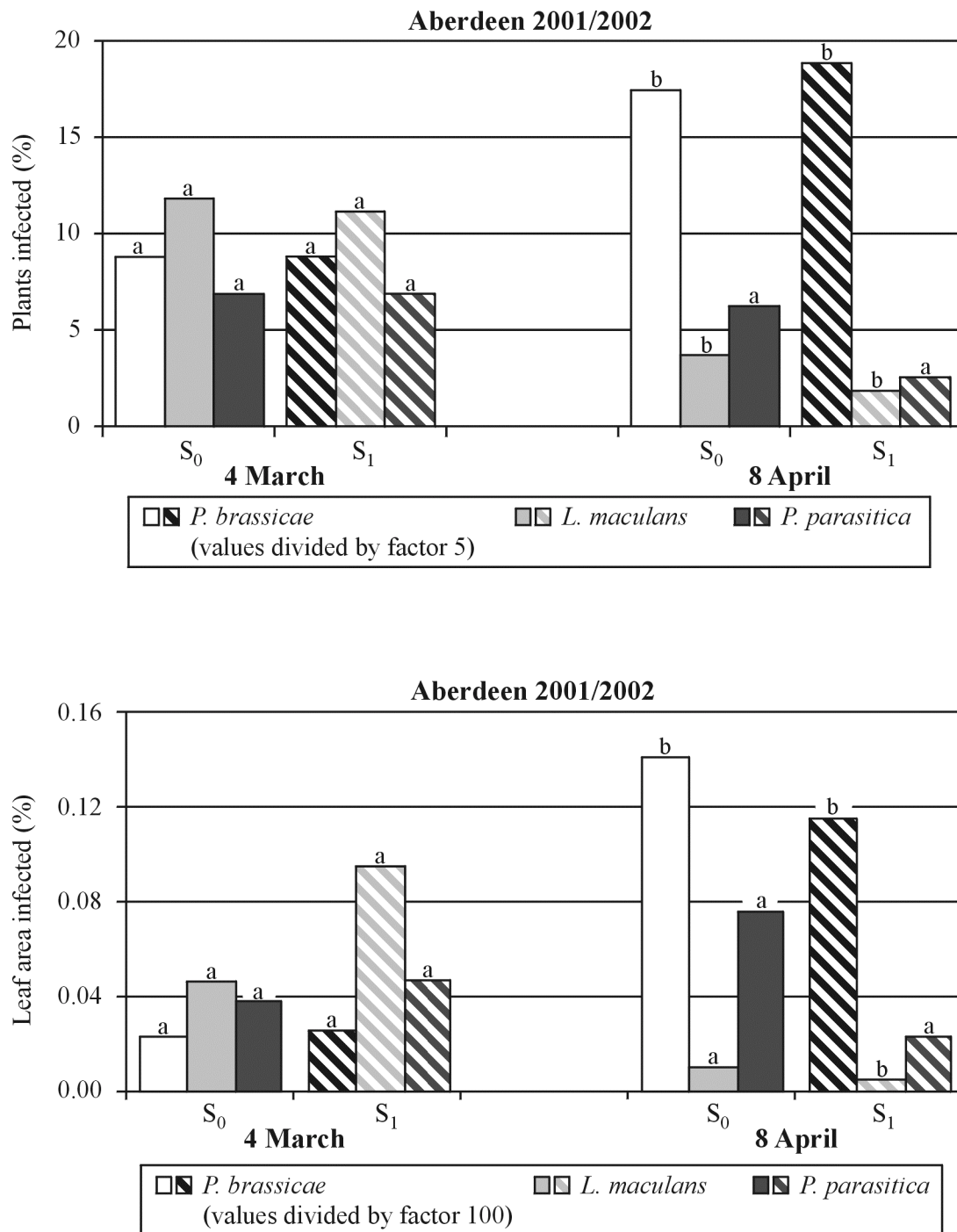


Figure 3.17: Effect of spring S application on the disease incidence and severity of fungal pathogens in Aberdeen (season 2001/2002) (non-fungicide treated plots; S fertilisation: 12 March; determined incidence and severity values for *P. brassicae* were divided by the factor 5 and 100, respectively; different letters denote significant differences between months at the 5% level by the t-test).

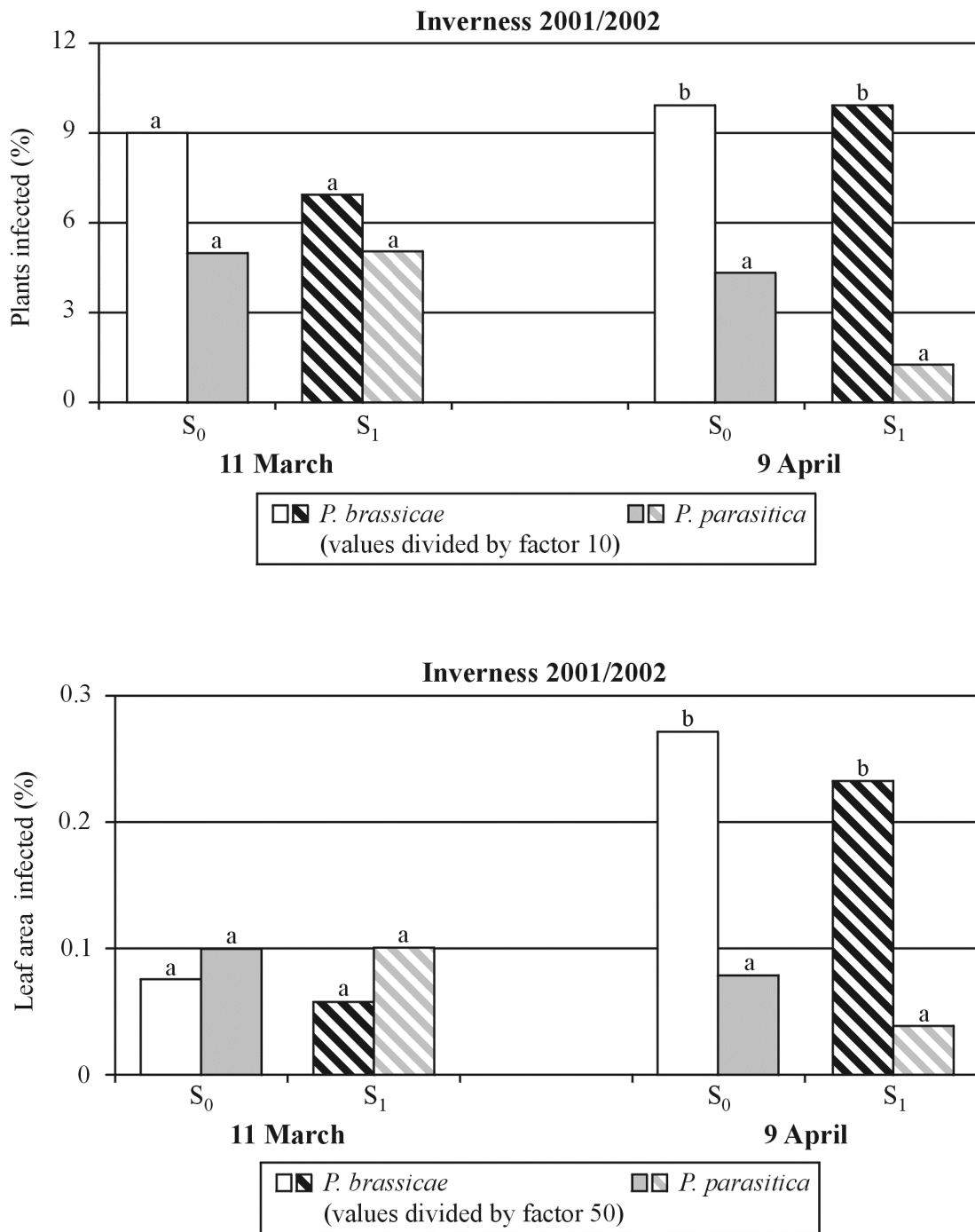


Figure 3.18: Effect of spring S application on the disease incidence and severity of fungal pathogens in Inverness (season 2001/2002) (non-fungicide treated plots; S fertilisation: 12 March; determined incidence and severity values for *P. brassicae* were divided by the factor 10 and 50, respectively; different letters denote significant differences between months at the 5% level by the t-test).

When one considers that at the time of leaf sampling (start of stem elongation) the collected leaves were affected by different fungal diseases, it is supposed that the leaf S metabolism is changed towards the synthesis of S-containing defence compounds in order to combat fungal attacks. Therefore, relationships between the disease incidence and severity of fungal pathogens and leaf S fractions were investigated. In Aberdeen in the second season, no correlations were found between fungal infections and leaf parameters (see Appendix: Table A.14 and A.19). On the other hand, in Inverness, the correlation analysis revealed negative relationships ($p < 0.05$) between the cysteine and γ -glutamyl-cysteine content in the leaf tissue with the disease severity of *P. brassicae* (Figure 3.19). This indicates that high concentrations of these metabolites might have some protective function. Cysteine and γ -glutamyl-cysteine are precursors of GSH and thus, elevated levels of these compounds may result in a higher GSH content. Based on these accounts, it might be possible that a high GSH content is also related with a low infection severity of *P. brassicae* even if correlations were not found in the present study. The lack of correlations can probably be accounted from the dependence of the GSH pool on a wide variety of factors.

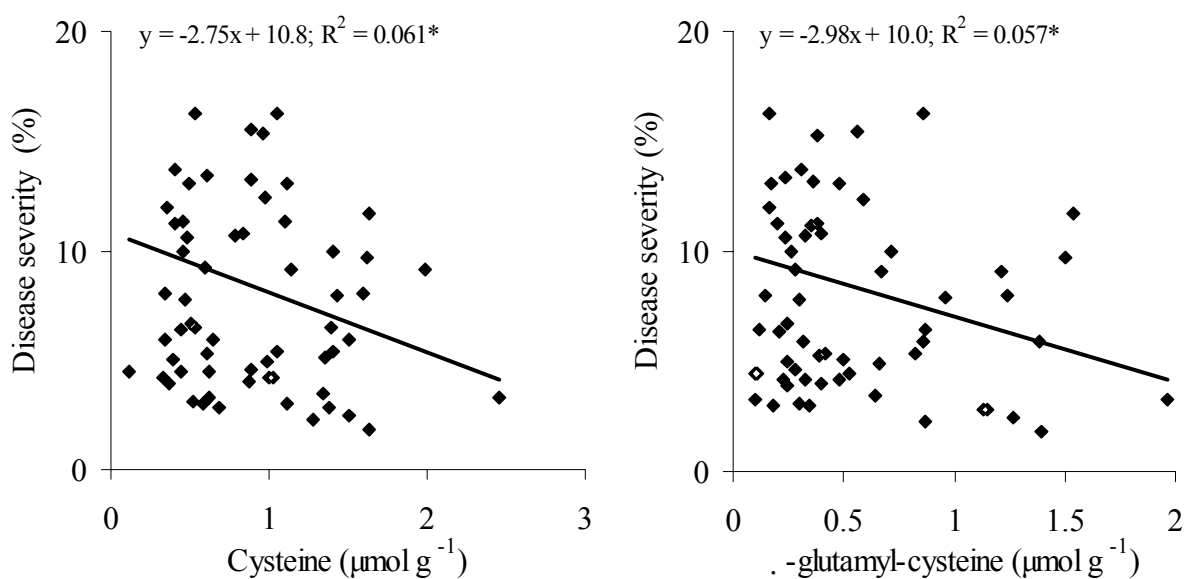


Figure 3.19: Relationship between the cysteine and γ -glutamyl-cysteine content in young leaves of winter oilseed rape with the disease severity of *Pyrenopeziza brassicae* in Inverness (season 2001/2002).

In summary, the results from the two Scottish sites revealed that the application of 100 kg S ha⁻¹ to winter oilseed rape, of which 50 kg S ha⁻¹ were applied in fall and 50 kg S ha⁻¹ were applied in spring, did not induce resistance towards *P. brassicae* within the two varieties tested at both sites. Nevertheless, negative correlations were found between the cysteine and γ -glutamyl-cysteine content in young leaves with *P. brassicae* infection severity, revealing a possible role of these metabolites in pathogenesis. The effect of S on *L. maculans* was unclear, while spring S application was effective against infections caused by the biotrophic fungus *P. parasitica*.

3.4 Changes in metabolite concentrations and enzymatic activities in response to *Pyrenopeziza brassicae* infection

As outlined in the previous chapter, *P. brassicae* was the most important disease in Scotland and was observed in Braunschweig in the second season as well. To have a better view on the metabolic changes that occur in plant tissues as a result of a fungal infection, leaf disc samples that showed visible symptoms and without visible symptoms of *P. brassicae* were taken from the upper third of the crop at the stage of high metabolic activity (beginning of stem elongation) from each plot in Braunschweig (season 2001/2002) and Aberdeen (season 2001/2002 and 2002/2003) (Figure 3.20).



Figure 3.20: Leaf disc sampling strategy for infections of winter oilseed rape leaves by *Pyrenopeziza brassicae* (photos: E Bloem).

In Aberdeen in the second season, beside infections of winter oilseed rape by *P. brassicae* (81% plants infected; 8.4% leaf area infected), additional infections caused by *L. maculans* (3% plants infected; 0.01% leaf area infected) and *P. parasitica* (13% plants infected; 0.2% leaf

area infected) were found at the time of sampling (see Appendix: Table A.19). In comparison, in the third season, only *P. brassicae* was observed in the crop at the sampling date (93% plants infected; 7.6% leaf area infected) (see Appendix: Table A.21). In Braunschweig, a scoring of infection by *P. brassicae* was not possible because of the high inconsistency of the infection rate and severity within individual plots, but at the time of sampling only white spore masses (Figure 3.20) – characteristic symptoms of *P. brassicae* – were present on leaves.

Since cysteine, GSH and H₂S are supposed to be the best candidates of *SIR* (Schnug et al., 1995a; Haneklaus et al., 2004), the aim of this part of the research work is to notice changes in the concentration of these metabolites in the vegetative tissue of winter oilseed rape in relation to infections caused by *P. brassicae*. Therefore, the influence of infections caused by *P. brassicae* along with the influence of S and N fertilisation, cultivar and fungicide treatment on the cysteine content, GSH level and enzyme activities putatively involved in the H₂S release (LCD and OAS-TL) has been determined. Experimental data were analysed by employing the multivariate GLM procedure and by correlation analysis. A one factorial ANOVA was carried out in the case of infections by *P. brassicae*. Individual data for the analysed leaf disc parameters are listed in the appendix (Table A.16 and A.17).

Since the fungicide treatment delivered no statistical differences with respect to the investigated parameters, its effect is not shown in this chapter (see Appendix: Table A.16 and A.17). First, the significance of the treatments for the content of individual plant parameters is presented, followed by bivariate correlation between individual S fractions, and between the analysed leaf disc parameters and the infection severity of *P. brassicae*.

The influence of S and N fertilisation, cultivar and infections by *P. brassicae* on the cysteine and GSH content in leaf discs of winter oilseed rape at the start of stem elongation at the experimental sites in Braunschweig and Aberdeen is summarised in Table 3.12 and 3.13, respectively. Evidently, the effect of cultivar, S and N fertilisation on these two parameters was similar as in the case of whole leaf samples, but the concentration of cysteine and GSH in leaf discs was found at lower levels compared to whole leaves (see Table 3.6 and 3.8).

Table 3.12: Influence of S and N fertilisation, cultivar and infections by *P. brassicae* on the cysteine and glutathione (GSH) content in leaf discs of winter oilseed rape at the start of stem elongation in Braunschweig.

Treatment	<i>Braunschweig</i>			
	<i>Cysteine</i>		<i>Glutathione</i>	
	$\mu\text{mol g}^{-1}$			
	<i>infected</i>	<i>non-infected</i>	<i>infected</i>	<i>non-infected</i>
S₀	1.01	0.46	13.2	11.0
S₁	1.70	0.70	15.8	11.5
N₁	1.30	0.59	14.0	10.1
N₂	1.40	0.57	15.0	12.4
<i>Bristol</i>	1.39	0.59	13.7	10.8
<i>Lipton</i>	1.31	0.57	15.3	11.6
LSD_{5%}	0.14	0.17	1.76	1.68
Treatment	<i>Cysteine</i>		<i>Glutathione</i>	
	$\mu\text{mol g}^{-1}$			
Infections by <i>P. brassicae</i>				
Yes	1.35		14.5	
No	0.58		11.2	
LSD_{5%}	0.14		1.27	

Table 3.13: Influence of S and N fertilisation, cultivar and infections by *P. brassicae* on the cysteine (Cys) and glutathione (GSH) content in leaf discs of winter oilseed rape at the start of stem elongation in Aberdeen.

Treatment	Aberdeen							
	2001/2002				2002/2003			
	infected		non-infected		infected		non-infected	
	Cys	GSH	Cys	GSH	Cys	GSH	Cys	GSH
	$\mu\text{mol g}^{-1}$							
S ₀	0.22	6.44	0.77	12.9	1.25	14.1	0.77	16.1
S ₁	0.43	10.4	0.88	14.5	1.42	15.2	0.92	15.5
N ₁	0.31	7.95	0.85	13.9	1.31	13.9	0.91	15.4
N ₂	0.34	8.86	0.80	13.4	1.36	15.4	0.79	16.2
<i>Bristol</i>	0.37	8.95	0.84	13.2	1.43	14.5	0.89	15.5
<i>Lipton</i>	0.28	7.86	0.81	14.2	1.24	14.8	0.80	16.1
LSD _{5%}	0.17	3.02	0.05	0.99	0.15	1.92	0.05	1.36
Treatment	2001/2002				2002/2003			
	Cys		GSH		Cys		GSH	
	$\mu\text{mol g}^{-1}$							
	Infections by <i>P. brassicae</i>	Cys		GSH		Cys		GSH
Yes	0.33		8.41		1.34		14.7	
No	0.83		13.7		0.85		15.8	
LSD _{5%}	0.09		1.63		0.08		1.12	

The analysis of variance showed that the application of S significantly increased ($p < 0.01$) the cysteine content in leaf discs of winter oilseed rape at the start of stem elongation at both sites (Table 3.12 and 3.13). In Braunschweig, the cysteine content increased by $0.7 \mu\text{mol g}^{-1}$ in infected leaf discs, whereas in non-infected leaf discs only an increase by $0.2 \mu\text{mol g}^{-1}$ was found (Table 3.12). In Aberdeen, the mean increase of the cysteine content was $0.2 \mu\text{mol g}^{-1}$ and $0.1 \mu\text{mol g}^{-1}$ in infected and non-infected leaf discs, respectively (Table 3.13). In Braunschweig and in the third season in Aberdeen, a higher cysteine content was found in infected leaf discs compared to non-infected leaf discs, whereas in the second season in Aberdeen the reverse pattern was noted (Table 3.12 and 3.13).

S fertilisation increased the GSH content in infected and non-infected leaf discs too, but the increase was not all the time significant (Table 3.12 and 3.13). Additionally, the GSH content showed large variations within the experimental field, indicating the presence of other factors that affect the foliar GSH levels (see Appendix: Table A.16 and A.17). In Braunschweig, S fertilisation increased the GSH content by $2.6 \mu\text{mol g}^{-1}$ and $0.5 \mu\text{mol g}^{-1}$ in infected and non-infected leaf discs, respectively (Table 3.12), whereas in Aberdeen the average increase was $2.5 \mu\text{mol g}^{-1}$ in infected leaf discs and $1.6 \mu\text{mol g}^{-1}$ in non-infected leaf discs (Table 3.13). Only at the experimental site in Braunschweig the GSH content increased with infection (Table 3.12).

N fertilisation had no effect on the cysteine and GSH content at both sites (Table 3.12 and 3.13). The analysis of variance also showed that differences between the resistant cultivar *Lipton* and the susceptible cultivar *Bristol* were not significant with respect to these two metabolites (Table 3.12 and 3.13). Therefore, both cultivars have the same potential to synthesise cysteine and GSH in the case of an infection by *P. brassicae*.

Infections by *P. brassicae* significantly increased ($p < 0.001$) the cysteine and GSH content in leaf discs of winter oilseed rape in Braunschweig (Table 3.12). The cysteine content increased by 2.3-fold when a visible infection was discovered, whereas the GSH content increased by 1.3-fold (Table 3.12). These findings indicate that both compounds were metabolised to a higher extent in leaf areas, which were obviously damaged by the pathogen. In Aberdeen, contradictory results were obtained (Table 3.13) and this was probably due to severe infections by *P. brassicae* and/or multiple pathogen infections observed during the experimentation on this site (see Appendix: Table A.19 and A.21). In the second season, a decrease of the cysteine and GSH content by 2.5-fold and 1.6-fold, respectively was found in leaf areas visually infected by *P. brassicae* (Table 3.13). In the third season, the cysteine content increased by 1.6-fold, whereas the GSH content decreased by 1.1-fold (Table 3.13).

The potential of oilseed rape to release H_2S by the enzymatic activity of LCD and OAS-TL was investigated at the experimental site in Braunschweig. These two enzymes are supposed to be responsible for the H_2S release from cysteine in higher plants (Burandt et al., 2001). LCD releases H_2S during cysteine degradation, while OAS-TL consumes H_2S during cysteine synthesis and free H_2S is only released in a side reaction. The influence of S and N fertilisation, cultivar and infections by *P. brassicae* on the LCD and OAS-TL activity in leaf discs of winter oilseed rape at the start of stem elongation is shown in Table 3.14. The LCD and OAS-TL activity showed large variations within the experimental field, indicating that the up-regulation

of the S metabolism depends on a wide number of environmental factors (see Appendix: Table A.16).

Table 3.14: Influence of S and N fertilisation, cultivar and infections by *P. brassicae* on the L-cysteine desulphhydrase (LCD) and O-acetylserine(thiol)lyase (OAS-TL) activity in leaf discs of winter oilseed rape at the start of stem elongation in Braunschweig.

Treatment	Braunschweig			
	LCD*		OAS-TL*	
	H_2S [nmol(mg protein x min) ⁻¹]		Cys [nmol(mg protein x min) ⁻¹]	
	infected	non-infected	infected	non-infected
S ₀	18.4	12.8	2460	2385
S ₁	16.4	10.4	2099	2069
N ₁	15.5	9.83	1970	1831
N ₂	19.2	13.4	2589	2623
<i>Bristol</i>	16.8	11.3	2216	2164
<i>Lipton</i>	18.0	11.9	2343	2290
LSD _{5%}	1.58	0.91	590	524
Treatment	LCD*		OAS-TL*	
	H_2S [nmol(mg protein x min) ⁻¹]		Cys [nmol(mg protein x min) ⁻¹]	
Infections by <i>P. brassicae</i>				
Yes	17.4		2279	
No	11.6		2227	
LSD _{5%}	1.16		372	

note: * data delivered by the Institute of Botany, University of Hannover, Germany

The analysis of variance revealed that S fertilisation significantly decreased ($p < 0.05$) the LCD activity in leaf discs of winter oilseed rape at the start of stem elongation (Table 3.14). The OAS-TL activity was not significantly influenced by S fertilisation, but also tended to be higher in the control plots (Table 3.14). S fertilisation decreased the LCD and OAS-TL activity by about 1.2-fold in both infected and non-infected leaf discs. Therefore, in plants that showed S deficiency (see Table 3.1), the activity of the cysteine synthesising and the activity of the

cysteine catabolising enzymes were higher than in plants, which were sufficiently supplied with S.

N fertilisation significantly increased the LCD and OAS-TL activity in leaf discs of winter oilseed rape (Table 3.14). In high N fertilised plots, an increase by about 1.2-fold and 1.4-fold for the LCD activity and by about 1.3-fold and 1.4-fold for the OAS-TL activity in infected and non-infected leaf discs, respectively was found.

The two cultivars, *Bristol* and *Lipton*, showed no significant differences with respect to the enzyme activities (Table 3.14). The activity of LCD and OAS-TL was only slightly higher for the resistant cultivar *Lipton*. Therefore, both cultivars seem to have the same potential to release H₂S in response to fungal infections.

Keeping in mind that OAS-TL is catalysing the cysteine synthesis and cysteine is the substrate for degradation by LCD, fungal infections can be expected to influence both enzyme activities. While the LCD activity significantly increased ($p < 0.001$) in the leaf tissue infected by *P. brassicae*, the OAS-TL activity increased only slightly and not significantly (Table 3.14). These findings suggest that in the case of infections caused by *P. brassicae*, LCD controls the H₂S release from leaves.

Correlation analysis

The results obtained from the correlation analysis between individual S fractions in leaf discs at the experimental site in Braunschweig are illustrated in Figure 3.21 (season 2001/2002). Highly significant positive correlations ($p < 0.001$) were found between the cysteine content with the γ -glutamyl-cysteine and the GSH content and between the γ -glutamyl-cysteine content and the GSH content in infected as well as in non-infected leaf discs (Figure 3.21). These findings reveal a strong dependence of the GSH synthesis on the cysteine and γ -glutamyl-cysteine pool. In Aberdeen, highly significant positive relationships were determined between the cysteine and GSH content in infected leaf discs, whereas no correlations were observed in non-infected leaf discs (see Appendix: Table A.17). There were no relationships between the cysteine, γ -glutamyl-cysteine and GSH content with the LCD and OAS-TL activity (Figure 3.21). A highly significant positive relationship ($p < 0.001$) between the LCD and OAS-TL activity was found in infected ($r = 0.683$) and non-infected leaf discs ($r = 0.485$) (Figure 3.21). Therefore, an increase in the OAS-TL activity was related to an increase in the LCD activity, revealing that the cysteine pool was maintained at low levels by the action of LCD.

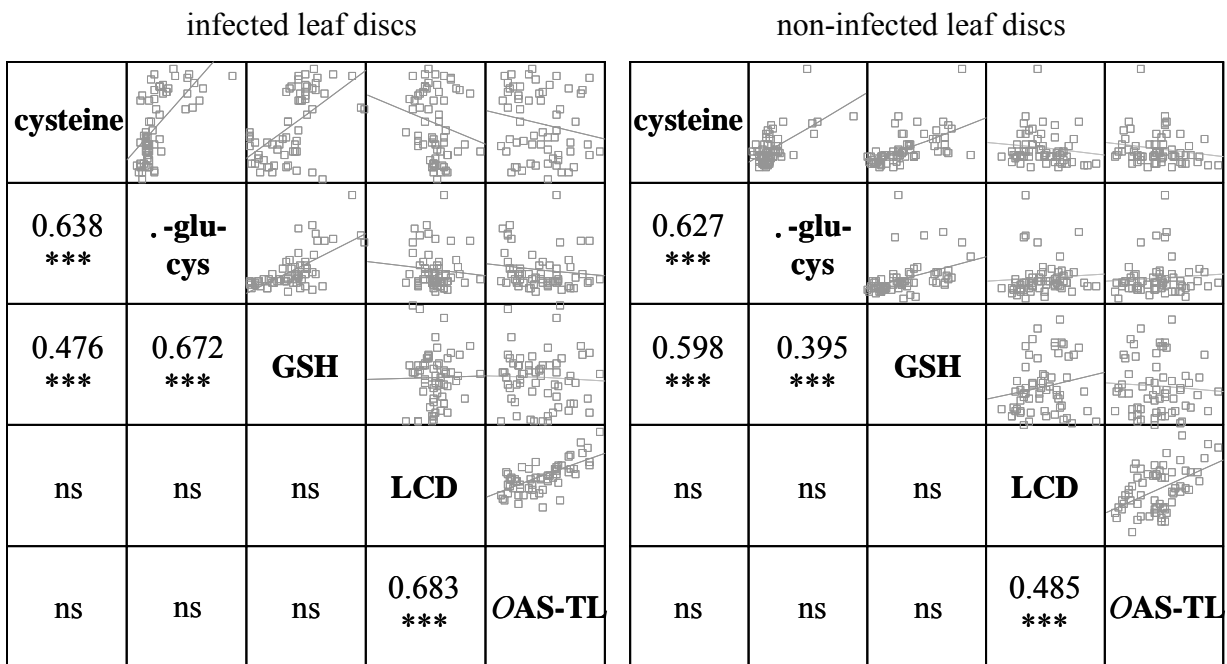


Figure 3.21: Correlation matrix for the cysteine, γ -glutamyl-cysteine (γ -glu-cys) and glutathione (GSH) content, and the L-cysteine desulphhydrase (LCD) and *O*-acetylserine(thiol)lyase (*OAS-TL*) activity in leaf discs of winter oilseed rape at the start of stem elongation in Braunschweig (season 2001/2002).

Previous investigations revealed strong correlations between the LCD and *OAS-TL* activity with the total S content (Burandt et al., 2001). N fertilisation has also proved to influence the enzyme activities (see above). Therefore, correlations between enzyme activities in leaf discs and the total S and total N content in whole leaves were also evaluated and the results are displayed in Figure 3.22. Weak, but significant negative relationships ($p < 0.05$) were found between the total S content and the LCD ($r = -0.223$) and *OAS-TL* ($r = -0.198$) activity (Figure 3.22). This indicates that under conditions of S deficiency both enzymes are activated. Contrary, the total N content was positively correlated with the LCD ($r = 0.392$; $p < 0.001$) and *OAS-TL* activity ($r = 0.209$; $p < 0.05$) (Figure 3.22).

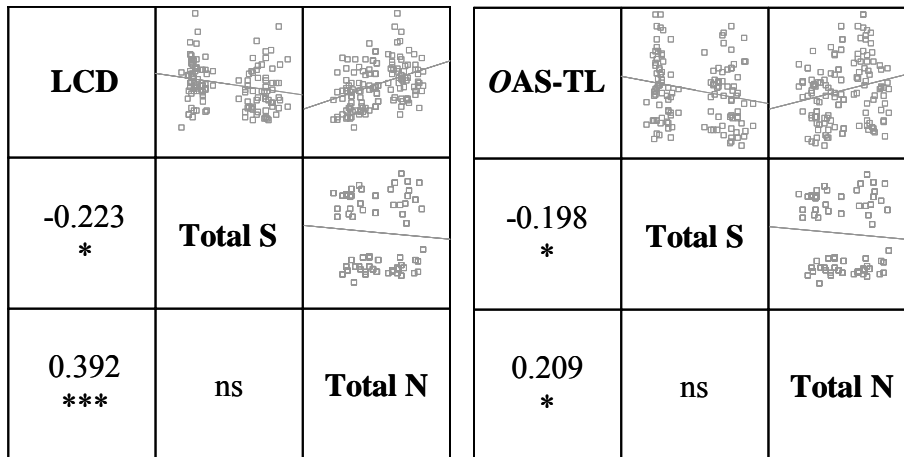


Figure 3.22: Correlation matrix for the L-cysteine desulphhydrase (LCD) and *O*-acetylserine(thiol)lyase (*OAS-TL*) activity in leaf discs, and the total S and total N content in young fully differentiated leaves of winter oilseed rape at the start of stem elongation in Braunschweig (season 2001/2002).

In Aberdeen, at the time of leaf discs sampling, a scoring of *P. brassicae* infection was carried out. Thus, the relationships between infections caused by *P. brassicae* and leaf disc S fractions were also investigated. In the second season no correlations were found (see Appendix: Table A.17 and A.19). The results derived from the correlation analysis for the third season are shown in Figure 3.23.

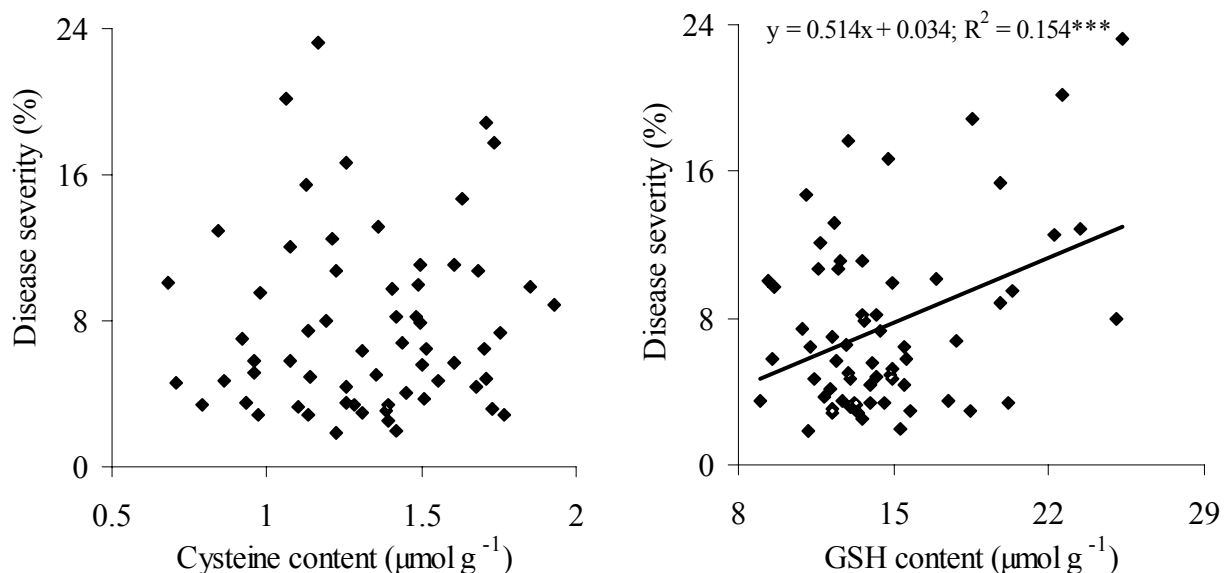


Figure 3.23: Relationship between the cysteine and GSH content in leaf discs of winter oilseed rape with the disease severity of *Pyrenopeziza brassicae* in Aberdeen (season 2002/2003).

The cysteine content was not correlated with the disease severity of *P. brassicae* (Figure 3.23). On the other hand, for the GSH content a highly significant positive correlation was found ($r = 0.392$; $p < 0.001$) (Figure 3.23). This might indicate that high GSH levels were required in plant tissues stronger infected by *P. brassicae* in order to keep the infection under control.

The results from the leaf disc analysis revealed that S fertilisation significantly increased the cysteine content. S had also a positive influence on the GSH content, though the increase was not all the time significant. On the other hand, N fertilisation had no effect on the cysteine and GSH content. Regarding the LCD and OAS-TL activity, S fertilisation had a negative effect, whereas N fertilisation increased both enzyme activities. The cultivars showed no marked differences with respect to the investigated parameters.

The results from the infection of winter oilseed rape leaves by *P. brassicae* showed marked differences between sites and seasons. This discrepancy was probably due to differences observed in the disease severity of *P. brassicae*. In the case of initial infections increased cysteine and GSH levels were found at the site of pathogen attack, whereas for severe infections a decrease was noted. Infections by *P. brassicae* influenced also enzyme activities: a significant increase was found for LCD, while the OAS-TL activity increased slightly and non-significantly.

Correlations were found between the cysteine, γ -glutamyl-cysteine and GSH content, but not between these compounds and the enzyme activities. The enzymes LCD and OAS-TL were positively correlated with each other and with the total N content and negatively correlated with the total S content. In the third season in Aberdeen, a positive relationship was found between the GSH content and the infection severity of *P. brassicae*.

3.5 Spatial patterns for total sulphur and sulphur-containing compounds in dependence on infections caused by *Leptosphaeria maculans*

In fall 2001 in Braunschweig, the infection of winter oilseed rape crop by *L. maculans* was observed and the infection severity was visually scored in the field at G.S. 14-15. Individual scoring data are listed in the appendix (Table A.18). Young, fully differentiated leaves of winter oilseed rape were taken from each plot at the beginning of stem elongation. Scoring of the fungal infection rate and plant sampling were carried out at different dates for the following reasons: S induced resistance is supposedly highest at a stage of high metabolic activity and should be distinctly expressed in young fully developed leaves in spring (Schnug and Haneklaus, 1998).

Metabolic modifications produced by *L. maculans* in the early growth stage, which are influenced by the S supply, should be therefore pronounced in the sampled plant tissue.

The aim of this part of the research work was to evaluate the spatial pattern for the risk of severe infections of winter oilseed rape by *L. maculans* and to assess the spatial variation of S-containing metabolites in dependence on the S supply. Geostatistical analysis was employed in order to head straight to this target, as a means to outline the impact and inter-relationships of unknown and given factors that influence the S nutritional status and the resistance of plants against fungal pathogens under field conditions. First, the results obtained by employing standard statistical procedures are shown, followed by the geostatistical analysis of the data.

The influence of S and N fertilisation, cultivar and fungicide treatment on the total S, GSH and GSL content was presented in chapters 3.1 and 3.2 (see Table 3.1, 3.6 and 3.7). The effect of the treatments on the infection severity of *L. maculans* was also evaluated by employing standard statistical procedures. In Table 3.15, the influence of cultivar and S fertilisation on the infection severity of *L. maculans* is summarised. The analysis of variance revealed no significant influence of S fertilisation on the infection severity (Table 3.15). On the other hand, data showed that the infection severity for the cultivar *Lipton* was significantly higher than the infection severity found for the cultivar *Bristol* (Table 3.15). These findings are in accordance with the predicted susceptibility of the cultivar *Lipton* to *L. maculans* (HGCA Recommended List WOSR 2003). The results of correlation analysis showed no relationships between the investigated S-containing compounds and the disease severity of *L. maculans* (see Appendix: Table A.9, A.13 and A.18). Therefore, by employing standard statistical procedures no involvement of GSH and GSLs in pathogenesis was revealed.

Table 3.15: Influence of cultivar and S fertilisation on the infection severity of *Leptosphaeria maculans* in winter oilseed rape crop (Braunschweig, fall 2001).

Variable/treatment	<i>S</i> fertilisation [kg ha^{-1}]		Cultivar		<i>LSD</i> _{5%}
	0	80 ²	<i>Bristol</i>	<i>Lipton</i>	
Infection severity ¹	4.18	4.21	3.73	4.66	0.36
note: ¹ scores for infection severity (6 = high; 1 = low); ² in total 150 kg S ha ⁻¹ ; the rest of 70 kg S ha ⁻¹ applied in spring					

It has been shown in the previous parts of this research work that the parameters investigated are highly dependent on a wide number of environmental factors (see chapter 3.1 and 3.2). Also within an experimental plot these factors may vary and therefore obscuring the effects caused by treatments. To include these spatial effects in the interpretation of the experimental results, plant physiological parameters and transformed score values for *L. maculans* infections were analysed for spatial variability using geostatistics. Geostatistics have been applied before in plant pathology for the analysis of the spatial variation of plant diseases, such as the evaluation of the spatial distribution and simulation of insect population (Ribes-Dasi et al., 2001) and the prognosis of the spreading of plant virus diseases (Nelson et al., 1994).

Spatial autocorrelations for plant parameters and *L. maculans* infections were analysed using the semi-variogram analysis. For the parameters total S and total GSL content no spatial correlations were found, which reveal that these parameters vary over shorter distances than the given lag distance. The experimental semi-variograms for GSH and the probability of severe *L. maculans* infections are illustrated in Figure 3.24. The results from variography are summarised in Table 3.16.

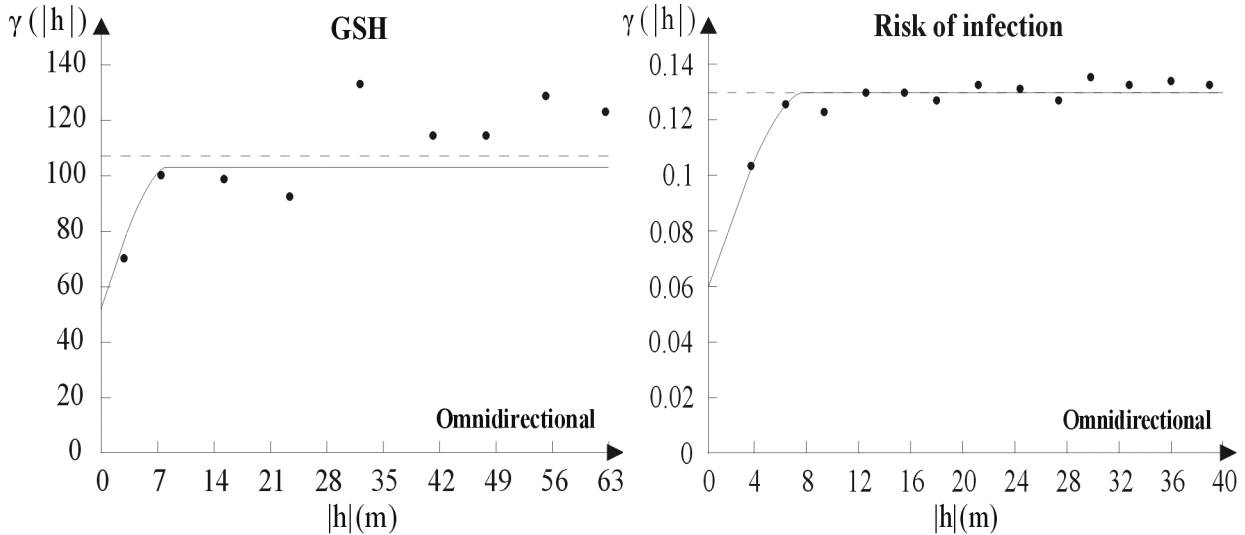


Figure 3.24: Omnidirectional semi-variograms for glutathione (GSH) and the risk of severe infections of winter oilseed rape by *Leptosphaeria maculans*.

Table 3.16: Semi-variogram parameters for glutathione and the probability of severe infections of winter oilseed rape by *Leptosphaeria maculans*.

Variable	Model	Nugget (C_0)	Sill ($C_0 + C_1$)	Range (a)	Nugget/Sill ($C_0 / C_0 + C_1$)
Glutathione	Spherical	52.2	102.2	8.26	0.51
Probability of severe infections by <i>L. maculans</i>	Spherical	0.06	0.13	7.60	0.46

A spherical model best fitted the probability of severe *L. maculans* infections and the leaf GSH content, but the nugget and sill values differed notably (Figure 3.24 and Table 3.16). The nugget value (C_0) in the semi-variogram was high for GSH (52.2), indicating a high fluctuation at the distance zero. Therefore, counts for closely spaced samples would be substantially different. In contrast, the semi-variogram for the probability of severe infections of winter oilseed rape by *L. maculans* had a small nugget value (0.06), which reveals that there is little small-scale random variability or unpredictability associated with this parameter. Thus, measurements of closely spaced samples would be almost identical. The sill value ($C_0 + C_1$) in the case of probability of severe *L. maculans* infections was small (0.13), indicating that the spatial autocorrelation between sample locations only occurs over distances much smaller than the plot dimension. In contrast, the high sill value for GSH (102.2) reveals that autocorrelations between sampling pairs exist over wide distances. The range value (a) represents the maximum distance up to which samples can be taken to build up a spatial image of the investigated parameter. For the parameters studied, this indicates that plant samples need to be taken in a distance not wider than 7 m to cover the spatial variability, as the range value for the variables GSH and the risk of high infection severity was 8.26 m and 7.60 m, respectively (Figure 3.24 and Table 3.16). The nugget/sill ratio (NR) provides an estimation of the random variation in the data and with NR values of 51% and 46% for GSH and the risk of severe fungal infections, respectively a moderate spatial dependency is revealed (Table 3.16).

Figure 3.25 reflects the small-scale spatial variability of the total S, GSH and total GSL content and the probability of severe infections of winter oilseed rape by *L. maculans* on the experimental field. The digital maps were created by interpolating the data for GSH by kriging, for total S and total GSL by employing the inverse distance procedure and for the risk of severe *L. maculans* infections by indicator kriging (Figure 3.25).

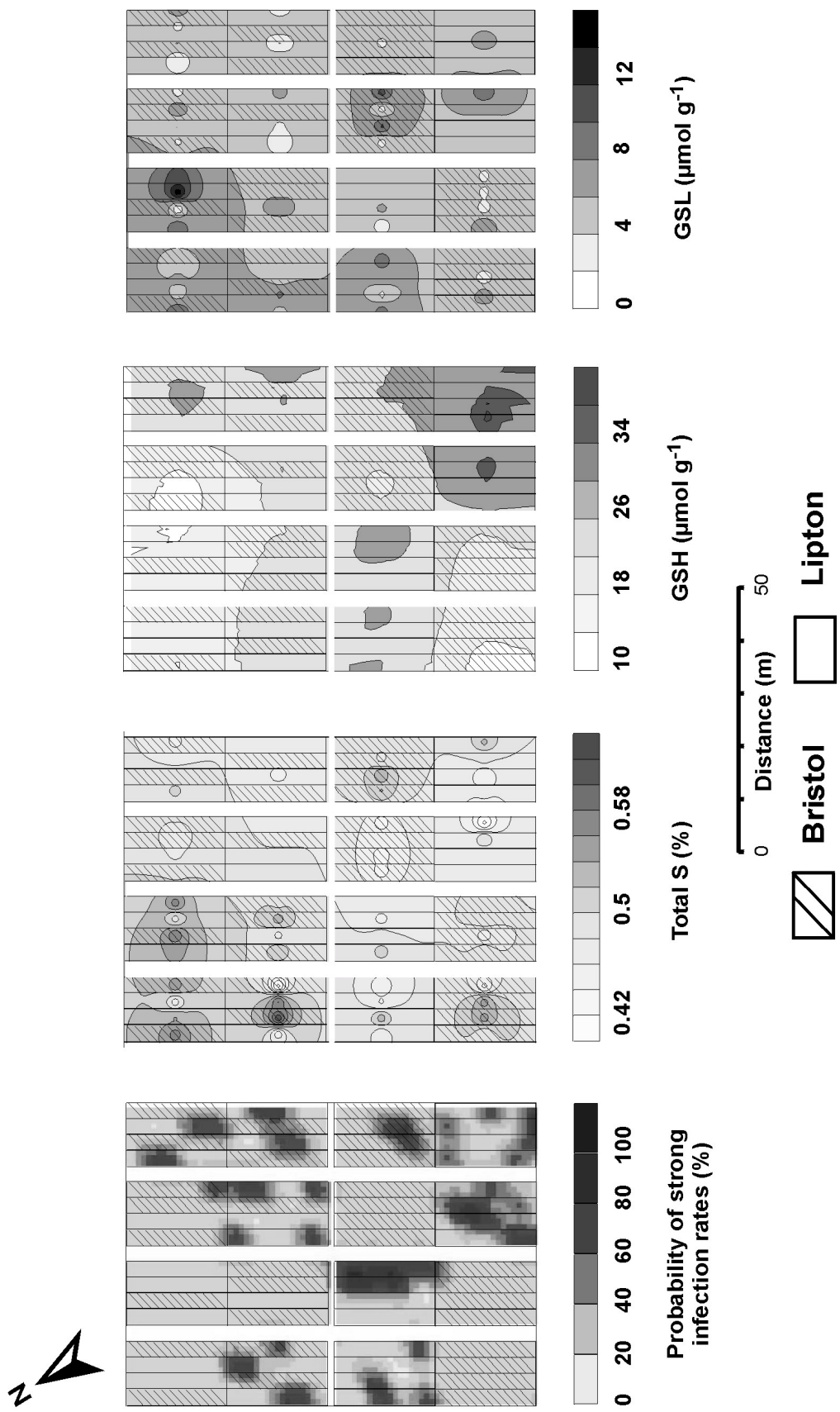


Figure 3.25: Small-scale spatial variability of the probability of severe infections of winter oilseed rape by *Leptosphaeria maculans*, the total S, glutathione (GSH) and total glucosinolate (GSL) content.

The probability of severe infections of winter oilseed rape by *L. maculans* differed widely on the experimental site with values from 20% to 60% (Figure 3.25). Infections were not homogenous throughout the field and in the south-eastern parts of the field the risk was distinctly higher than in the northern and western areas (Figure 3.25). In general, the risk of severe infections by *L. maculans* was higher for the cultivar *Lipton*. On plots where *Lipton* was grown, a high risk of severe infections coincided with increased GSH levels in the vegetative plant tissue. Such relationships were less pronounced for the cultivar *Bristol*. By comparison, spatial patterns for total S and total GSLs revealed no significant differences between cultivars (Figure 3.25).

In an attempt to determine relationships between *L. maculans* infection and plant parameters and to understand the specific factors responsible for fungal distribution patterns, the distribution of plant parameters was compared with the spatial distribution of *L. maculans*. Therefore, overlaying the maps, areas with a high GSH content matched those with a high probability of severe fungal infections, while inverse relationships were found for the total S and total GSL content (Figure 3.25). Apparently, plants responded to fungal infections with an increased GSH synthesis. There was no consistent influence of S fertilisation on the GSH content (see Table 3.6), so it can be assumed that infections by *L. maculans* caused an increased GSH synthesis in plants.

The fact that a low S nutritional status occurred at the same locations where an enhanced risk of severe infections by *L. maculans* was found indicates that an insufficient S supply results in the loss of plant vigour and promotes therefore the sensitivity of plants to fungal infections. The significance of the concurrence of low GSL content and a high risk of fungal infections remains obscure, but suggests that a higher GSL content had some protective function.

The results from the geostatistical analysis revealed that the probability of severe *L. maculans* infections varied spatially and the risk of a strong infection was higher for the cultivar *Lipton* than for the cultivar *Bristol*. The spatial pattern for the probability of severe *L. maculans* infections matched that of the S status of the crop in such a way that a higher risk was related to a lower S status and a lower GSL content. An inverse pattern was found for the GSH concentration.

4 Discussion

The main objective of this research work was to investigate the effect of the S nutrition on different S-containing metabolites in relation to fungal infections. Field experiments were based on the hypothesis that the S nutrition of plants enhances biochemical processes in the primary and secondary S metabolism and thus, it may induce resistance against fungal pathogens (Schnug et al., 1995a). To reach this goal it was necessary to choose sites, which are potentially S deficient and show a high infection risk for fungal pathogens. Scotland is a country with extremely low atmospheric S depositions (Dore et al., 2003) and with a very high infection pressure for *P. brassicae* (Lacey et al., 1987). Based on these accounts, two field trials were conducted on two sites in Scotland and the third one in an area with low infection pressure for *P. brassicae* and moderate atmospheric S depositions in Braunschweig (Germany). The relatively high S requirement of oilseed rape and its considerable importance for agriculture in northern Europe made this crop ideal to investigate the role of S-containing metabolites in plant resistance mechanisms in order to exploit them as a means of resistance to pathogens. In these experiments, the S supply of oilseed rape was provided by the application of sulphate to the soil and thus avoiding the potential direct fungicidal effect that may have been associated with foliar applied S.

In the present work, attention has been focussed on cysteine, GSH, GSLs and the release of H₂S. These S compounds are supposed to be the best candidates for *SIR* because of their direct dependence on S. Other compounds considered to be an active part of the induced multiple plant defence mechanisms against microbial pathogens are phytoalexins (Sinha, 1995). Ingham (1973) defines phytoalexins as “antibiotics formed in plants via a metabolic sequence induced either biotically or in response to chemical or environmental factors”. According to this definition, the formation of elemental S, the stress induced formation of pathogenesis-related proteins (PR proteins) and a novel class of low-molecular weight antibiotics come under the term phytoalexins (Haneklaus et al., 2004). The involvement of low-molecular weight antibiotics in the plant resistance is yet not fully understood since only few investigations have demonstrated that the timing, the location of synthesis and their accumulation is directly associated with specific sites of pathogen perturbation (Hammerschmidt and Nicholson, 2000). Their involvement in *SIR* is even more obscure and can only be speculated from the dependency of their precursors on S (Schnug et al., 1995a; Haneklaus et al., 2002 and 2004). Regarding PR proteins, the positive relationship of high levels of PR proteins with enhanced disease resistance in plants has been documented in many, but not all cases (Bohlmann, 1999; Punja, 2001) and their possible role in *SIR* still requires empirical proof. It is the aim of the German Plant S group

(Teilprojekt 2 – Rüdiger Hell, Heidelberg) to investigate the role of PR proteins in the defence mechanisms of plants.

The discussion of the results of this thesis starts with the evaluation of the S and N supply, cultivar and fungicide treatment on mineral composition (chapter 4.1) and S-containing primary and secondary compounds (chapter 4.2). In the following chapter, the importance of the S nutrition for plant resistance is considered along with a discussion of signal transduction pathways that plants activate in order to induce specific responses to different types of fungi (chapter 4.3). In the next two chapters, the significance of S-containing metabolites for crop resistance is discussed (chapter 4.4 and 4.5). As geostatistical methods have never been used to investigate the relationships between plant parameters and fungal diseases, their suitability to do so, based on the results of this work, are evaluated (chapter 4.5). The chapter is completed with a discussion on the significance of the present contribution for the identification of metabolites and processes that trigger *SIR* (chapter 4.6).

4.1 Relationship between S supply, productivity and mineral nutrient status

A special problem of growing double low oilseed rape is that these varieties are more sensitive to a low S supply than the old single low varieties (Schnug, 1989). Therefore, double low varieties require S fertilisation in order to improve the protein content and to ascertain sufficient yields. Previous investigations have shown substantial yield responses to S fertilisation on oilseed rape in the UK (McGrath and Zhao, 1996), Scotland (Booth et al., 1991) and Germany (Schnug, 1987; Haneklaus et al., 1999). Field trials carried out in the UK showed that yield was increased 10% to 327% by S applications (McGrath and Zhao, 1996). In Scotland, a significant yield increase was reported in three oilseed rape varieties when 32 kg elemental S ha⁻¹ was applied (Booth et al., 1991). Field experiments conducted in Germany also showed that fertilisation with elemental S or sulphate significantly increased the seed yield of oilseed rape (Schnug, 1987; Haneklaus et al., 1999).

Nevertheless, the responsiveness of oilseed rape to S is not universal. This is because S is a geogenic abundant element (Clark, 1979) compared for instance with N, the origin of which in agro-ecosystems is predominantly anthropogenic. High amounts of S are found in the atmosphere in regions closed to industrial areas and here atmospheric S represents an important source of S for plants. Vast amounts of S are bound in minerals (e.g. gypsum and pyrite) and can be delivered by the ground water to the surface (Schnug and Haneklaus, 1998). Eriksen et al. (1998) reported that the soil water and shallow ground water have much higher sulphate

concentrations than the water of precipitation, which is due to the natural S sources in the ground or to the charging by atmospheric sources. Therefore, under field conditions, crop responses to S fertilisation vary largely according to site, season, the form of S and varietal type. Comparative trials conducted in Scotland by Booth et al. (1991) indicated the importance of site in terms of predisposition to S deficiency and of varietal type in terms of response to S.

In the present study, oilseed rape plants showed S deficiency on all three experimental sites (see Table 3.1) and therefore yield responses to S fertilisation were expected. However, the present experiments did not give the expected significant yield increases to the application of S, whereas N fertilisation and fungicide treatment proved to have a significant positive effect (see chapter 3.1). In other field experiments conducted on S-deficient sites in the UK, the seed yield was also not significantly increased by the addition of up to 80 kg S ha⁻¹ (Blake-Kalff et al., 2000). Field experiments by Donald et al. (1993), carried out with the variety *Rafal*, showed no effect of S fertilisation on seed yield as well, while an increase of the yield by N fertilisation was found. The lack of significant yield response to the application of S observed in the present study might be due to a number of factors, which include:

- a) a delay in the utilisation of S, so that the S supply did not affect the initial development of seeds;
- b) an insufficient supply of other macro and/or micro-nutrients, so that oilseed rape plants could not efficiently react to S fertilisation; Liebig's "Law of the Minimum" (1855) states that the exploitation of the genetically fixed yield potential of crops is limited by a variable, which is insufficiently supplied to the greatest extent; for instance, in the third season in Aberdeen, oilseed rape crop was deficient in K (see Appendix: Table A.10);
- c) the presence of severe weather conditions (see Appendix: Table A.1 to A.3);
- d) the infection of winter oilseed rape crop by fungal pathogens (see Appendix: Table A.18 to A.21);
- e) the input of S from other sources such as mineralization, atmospheric inputs (estimated moderate in Braunschweig; see Table 2.1), soil (high soil S status in Aberdeen and Inverness; see Table 2.3 and 2.4) and possibly the subsoil.

Nevertheless, the seed yield increased slightly and in Figure 4.1 the relative yield increase in dependence on S fertilisation and per each kilogram of S applied to the soil is shown. The results revealed that the highest yield response to S fertiliser applications was in Inverness (approximately 8% / 100 kg S ha⁻¹ and 0.08% / kg S ha⁻¹; Figure 4.1), where atmospheric S

depositions were rated very low (Dore et al., 2003). These results confirm the findings of Schnug and Haneklaus (1994) that the poorer a site in S the stronger the response to the S supply.

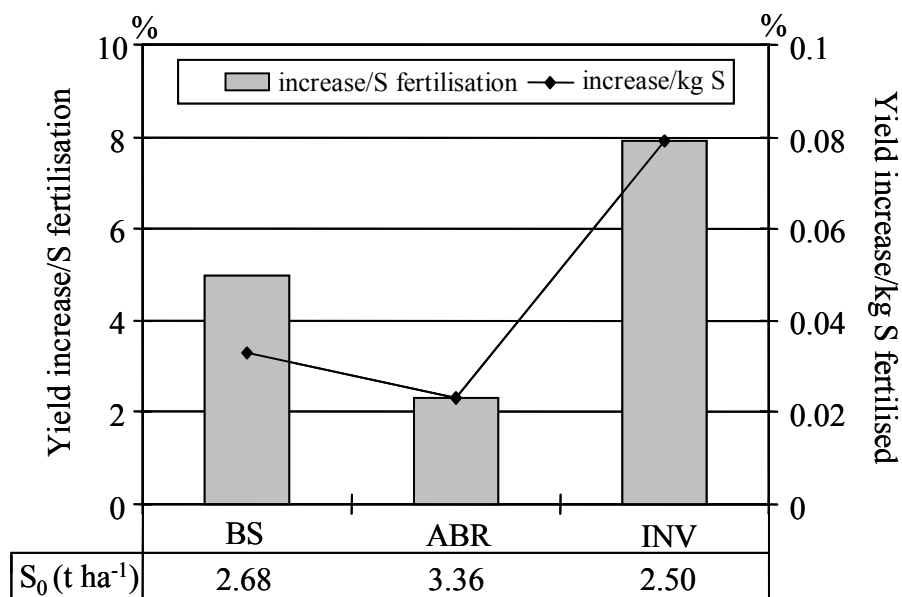


Figure 4.1: Relative seed yield increase in dependence on S fertilisation and per kg S applied ha⁻¹ in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (mean over three years: 2000-2003; relative increase in relation to control plots).

Schnug (1988) has established a relationship between the total S content in young fully developed leaves of oilseed rape and the relative seed yield. Based on this investigation, the expected yield can be calculated from the analysed total S content in young leaves when only S is considered yield limiting. It has been therefore reported that above total S concentrations of 6.5 mg S g⁻¹ in the foliar tissue of oilseed rape no further yield increases by increasing tissue S concentrations are to be expected (no effect values) (Schnug, 1988; Schnug and Haneklaus, 1994). When one considers that in the present work S fertilisation increased the total S content in young leaves above the critical nutrient threshold of 6.5 mg S g⁻¹ (see Table 3.1), an optimum growth and subsequently maximum yield should have been reached. Since yields recorded were poor (about 3 t ha⁻¹), this indicates that other factors were yield limiting. For instance, the poor seed yields recorded in the second season on all three experimental sites (see Table 3.1) might be due to the high levels of precipitation falling in July 2002 (see Appendix: Table A.1 to A.3). Because of these severe weather conditions, the harvest had been delayed. Another factor that may have a negative impact on yield is the infection of winter oilseed rape by fungal pathogens (see Appendix: Table A.18 to A.21). High yield losses were reported in the last decades in

Scotland as a result of infections caused by *P. brassicae* (Fitt et al., 1997 and 1998). The mechanisms of yield loss due to *P. brassicae* infections are not clear and also the yield formation in oilseed rape is poorly understood. However, work funded by HGCA has shown that for every 10% of plants infected by *P. brassicae* at green bud stage there is a potential of 0.14 t ha⁻¹ yield loss (Sutherland, 2001). The fact that the cultivar *Lipton* yielded more than the cultivar *Bristol* is well in accordance with the predicted susceptibility of *Bristol* to *P. brassicae* (Gladders et al., 1998).

Regarding the mineral content, there was a significant effect of S applications on the total S and SO₄-S content. In Figure 4.2, the relative increase of the total S and SO₄-S content in leaves and of the total S content in seeds is shown (season 2001/2002). It can be therefore seen that at the Inverness site the highest responses to applied S, expressed in terms of its concentration in leaves and seeds, were recorded, which could be again attributed to the very low atmospheric S depositions in this region (Figure 4.2).

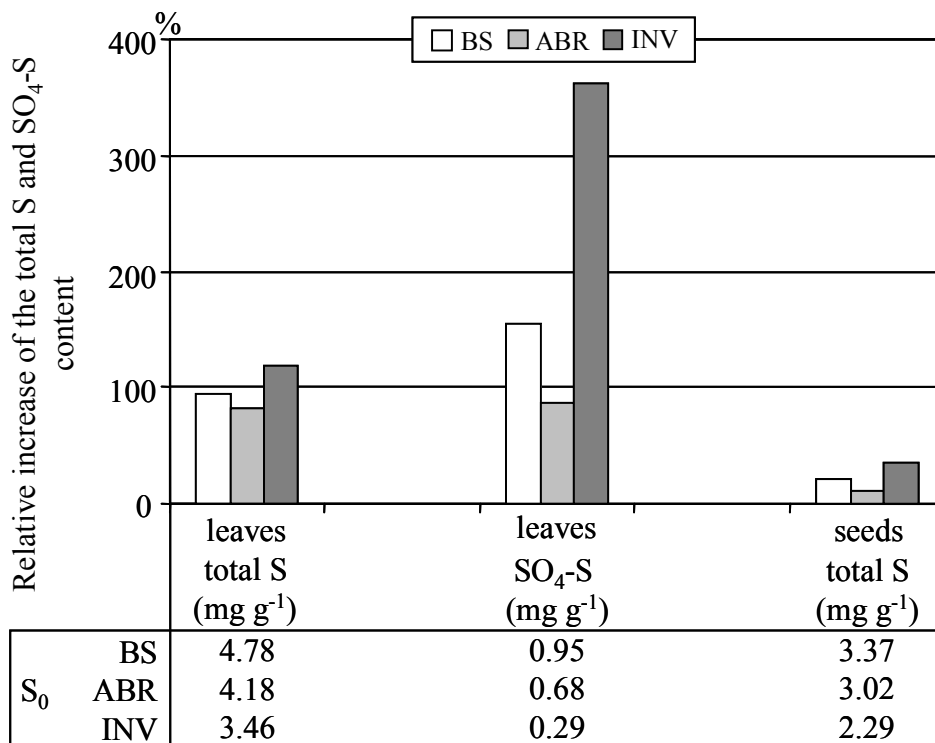


Figure 4.2: Relative increase of the total S and SO₄-S content in young leaves of winter oilseed rape at the start of stem elongation and of the total S content in seeds in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (relative increase in relation to control plots).

The seasonal variability noticed in the present work regarding the content of mineral nutrients (see Table 3.5) might be due to technical factors of production (e.g. the timing of S applications) as well as factors affecting the physical mobility and losses of S from soils. Previous investigations showed that leaching of sulphate depends on the soil type, the amount of winter precipitation and the concentration of $\text{SO}_4\text{-S}$ in the soil water during the leaching period (Bloem, 1998). The significance of soil physical properties, especially soil texture, for the S supply of crops is discussed by Bloem (1998). The author showed that on sandy soils a winter precipitation between 390-560 mm lowers the concentration of sulphate in a depth of 90 cm to approximately 3 mg kg^{-1} . From the meteorological data it was noticed that fall-winter precipitation rates were high during the experimentation period (see chapter 2.1 and Appendix: Table A.1 to A.3). This indicates that sulphate was highly susceptible to leaching during this period. Since the distribution of precipitation varied between the experimental seasons (see Appendix: Table A.1 to A.3), the S supply and demand differed also greatly. Therefore, the discrepancy between seasons observed for the total S and $\text{SO}_4\text{-S}$ content as well as for the other investigated S fractions (see later on) can be explained to a great extent by the seasonal variation in weather conditions. Schnug and Haneklaus (1998) also indicated that a maximum of 16% of the variability of S concentrations in leaves could be explained by the variability of available S in soils. Other factors responsible for the seasonal variability of plant parameters might be: different developmental stage of plants at the time of sampling and different degrees of fungal infections during the experimentation (see chapter 3.3, 3.4 and 3.5).

N fertilisation has proved to have a positive effect on the total N content in young leaves of winter oilseed rape at the start of stem elongation (see Table 3.4). At the experimental site in Braunschweig, the seasonal variation of the total N content in the leaf tissue can be explained by different timings of the second N application. In the second and third season, the second N application occurred after leaf sampling (see Appendix: Table A.6). A different extent of leaching can also explain the seasonal variability of the total N content in leaves, as both sulphate and nitrate are prone to leaching (Schnug, 1997).

Sulphur is known to interact with almost all essential macronutrients, secondary nutrients and micronutrients (Abdin et al., 2003). These interactions can either enhance or reduce growth and yield of crops by influencing the nutrient uptake and utilisation. S and N relationships in terms of crop yield and quality have been established in many studies, as both elements are needed for protein synthesis (Lakkineni and Abrol, 1992; Schnug et al., 1993b). For instance,

McGrath and Zhao (1996) observed an increase of 42-267% in the seed yield of *B. napus* with the application of 40 kg S ha⁻¹ together with 180 and 230 kg N ha⁻¹. Work of Booth et al. (1991) with rapeseed showed that there was a significant interaction between S and N and that the maximum yield was obtained when S and N applications were balanced. In the present work, no interactions between S and N were found with respect to the seed yield. This was probably due to the fact that S had no significant effect on yield and therefore interactions between S, N and yield were hardly to expect. However, high N rates resulted in a higher total S content in seeds and this indicates that the utilisation of N and S are dependent upon one another via the metabolism of amino acids.

Indirect calculation of the total S content in rapeseed

The total S content in rapeseeds can be calculated from the total GSL content in seeds following the X-RF conversion algorithm (see chapter 2.4.1) (Schnug and Haneklaus, 1990). In Table 4.1, the results obtained from the indirect calculation of the total S content in seeds and the results from the analysis of the total S content by means of ICP-AES are shown. As can be seen, the calculated total S contents are slightly lower than the concentrations determined by the ICP-AES analysis (Table 4.1). This might be due to the fact that glucosinolates, which are originally in seeds are decomposed during on-column desulphatation, which requires several hours reaction time (Moller et al., 1985). However, the total S content analysed by ICP-AES correlates closely ($p < 0.001$) with the total S content derived from the indirect calculation (Figure 4.3). Previous investigations also showed that the X-RF method provides a precise, rapid and simple determination of the total GSL content in rapeseeds (Schnug and Haneklaus, 1988 and 1990). It can be therefore concluded that the X-RF conversion algorithm can be successfully applied when one of the parameter is known (i.e. total S or total GSL content), avoiding the costs for further analysis.

Table 4.1: Total S content in seeds of winter oilseed rape analysed by ICP-AES and recalculated by means of X-RF non-linear calibration method.

Treatment	<i>Total S</i>					
	<i>mg g⁻¹</i>					
	<i>ICP-AES</i>			<i>Indirect calculation (X-RF)</i>		
	<i>BS</i>	<i>ABR</i>	<i>INV</i>	<i>BS</i>	<i>ABR</i>	<i>INV</i>
<i>2000/2001</i>						
S₀	3.20	2.76	2.29	2.81	2.60	1.98
S₁	3.58	2.98	2.87	3.21	2.88	2.72
<i>2001/2002</i>						
S₀	3.37	3.02	2.29	3.19	2.63	2.19
S₁	4.10	3.36	3.08	3.73	2.94	3.09
<i>2002/2003</i>						
S₀	2.88	2.90	3.04	2.44	3.01	2.87
S₁	3.65	3.16	3.37	3.21	3.29	3.19

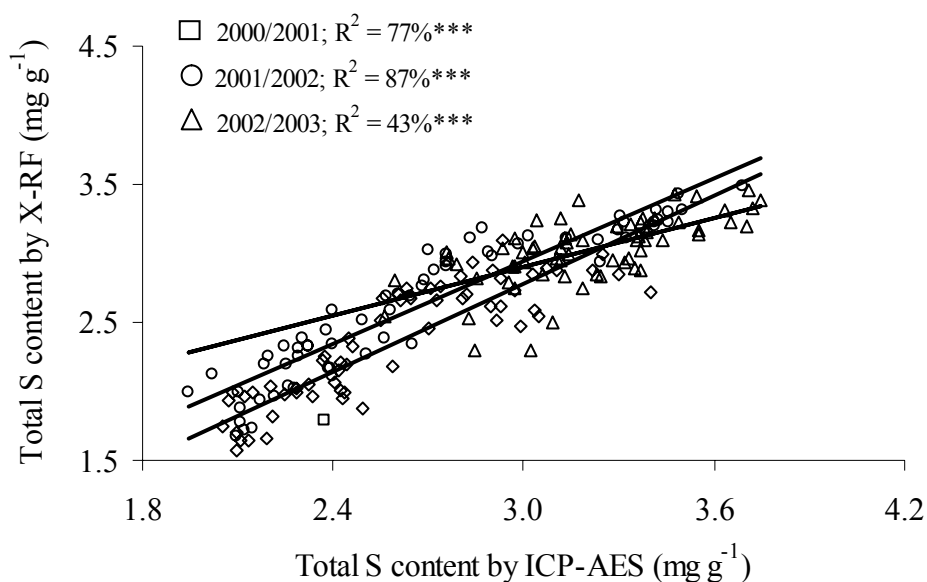


Figure 4.3: Relationship between the total S content in seeds of winter oilseed rape analysed by ICP-AES and the total S content derived from the indirect calculation (X-RF method, non-linear calibration) (Inverness).

From the points discussed above more evidences are delivered that with S fertilisation mineral S fractions can be increased. Factors such as site, season and variety have been also proved to influence the S uptake. It is supposed that S, site, season and varietal type are also the main factors influencing the organic S content of winter oilseed rape plants. Therefore, in the next chapter, the significance of these factors together with the significance of the N supply for the content of S-containing primary and secondary compounds, based on the results of this investigation, is discussed.

4.2 Significance of cultivar, sulphur and nitrogen fertilisation on sulphur-containing primary and secondary compounds of winter oilseed rape with view to SIR

Cysteine is the first stable organic S compound in the plant S metabolism. From cysteine several S-containing biochemical metabolites are derived among which GSH and GSLs have received special attention in the present study because of their putative involvement in *SIR* (Haneklaus et al., 2002 and 2004). Previous investigations showed a positive influence of the S supply on the concentration of S-containing metabolites in plant tissues (De Kok et al., 1981; Schnug et al., 1995b; Schnug, 1997; Haneklaus et al., 1999; Wielebski et al., 1999). The present results also revealed that S applications have a strong and significant influence on the cysteine, GSL and protein-S content (see chapter 3.2). With a better S supply the plant vigour is improved, elevated levels of S metabolites are synthesised and therefore the potential of *SIR* is increasing. On the other hand, no significant influence of S fertilisation on the GSH content in the leaf tissue was found: in some cases the GSH content was higher with S applications, while in others a reverse pattern was observed (see Table 3.6). Moreover, the GSH concentration in the foliar tissue showed a high variability and ranged from 11 $\mu\text{mol g}^{-1}$ to 30 $\mu\text{mol g}^{-1}$ (see Appendix: Table A.13 to A.15). These levels are on average 25 times higher than the GSH concentrations found in vegetative tissues of oilseed rape in experiments conducted under controlled conditions by Schnug et al. (1995b) or the ranges reported in spinach leaf tissues by De Kok et al. (1981). The reason for this discrepancy may rely on different rates of S fertilisation and growth conditions of plants in pot and field trials and also stresses the dependence of the GSH concentration on environmental factors. Tausz et al. (2003) reported that under field conditions the GSH concentration in foliar plant tissues might be influenced by a wide variety of abiotic factors such as light, drought, temperature, exposure to heavy metals, herbicide, and atmospheric pollution. Additionally, the present results revealed the dependence of the GSH pool on biotic stress, namely fungal infections (see chapter 3.4 and 3.5). In chapters 4.4 and 4.5, the changes observed in the foliar GSH content as a result of fungal infections are discussed. The

biochemical mechanisms through which the external factors modulate the foliar content of GSH are not known in details, but these “candidates” are altered the rates of synthesis, degradation, import and export (Noctor et al., 1997). Because of this dependence of the GSH content in plants on various biotic and abiotic factors, under natural conditions a strong influence of the S supply on the GSH content is hardly to occur.

The effect of varying availability of several nutrients (N, P, S) on GSLs contained within vegetative parts of the plant was investigated as early as the 1940's (Pryor, 1940; Schnug, 1989; Booth et al., 1991). The findings of these experiments, showing that S applications increased the GSL levels in vegetative and generative tissues (see Table 3.7, Figure 3.4 to 3.6), are in accordance with previous investigations (Schnug, 1989; Booth et al., 1991). The seasonal variation of the GSL levels within the study sites (see Table 3.10) might be explained to a great extent by the seasonal variability of the S supply (see chapter 4.1), whereas the regional variability of the GSL contents (see Table 3.10) corresponds with the regional variability of atmospheric S depositions (see Table 2.1).

Over three years research period, the mean total GSL content in young leaves was $3.6 \mu\text{mol g}^{-1}$ when no S was applied and $4.7 \mu\text{mol g}^{-1}$ when $100/150 \text{ kg S ha}^{-1}$ were applied. The corresponding values for seeds were $6.2 \mu\text{mol g}^{-1}$ and $9.9 \mu\text{mol g}^{-1}$, respectively. In experiments investigating the effect of S on GSLs in oilseed rape from the vegetative stage to maturity, Booth et al. (1991) reported ranges for the total GSL content of the cultivar *Cobra* between $7.8 \mu\text{mol g}^{-1}$ to $14.1 \mu\text{mol g}^{-1}$ in vegetative tissues at the start of stem elongation and between $12.2 \mu\text{mol g}^{-1}$ to $17.1 \mu\text{mol g}^{-1}$ in seeds. Therefore, the GSL levels found in the present study, in both leaves and seeds, are rather low when compared to the field experiments of Booth et al. (1991). These differences may be due to genotypical differences between cultivars used in these experiments and/or different experimental and environmental conditions.

In the present work, the increase in the content of total GSLs was mostly due to an increase in the content of alkenyl GSLs, mainly represented by progoitrin, gluconapin and glucobrassicinapin. These findings are in line with the results of Schnug (1988) and Zhao et al. (1994). Alkenyl GSLs are derived from methionine, which itself has been found to be influenced by S availability (Schnug, 1997). The figures 4.4 and 4.5 show the relative increase of the individual and the total GSL content in young leaves and seeds, respectively as a result of S fertilisation (season 2001/2002).

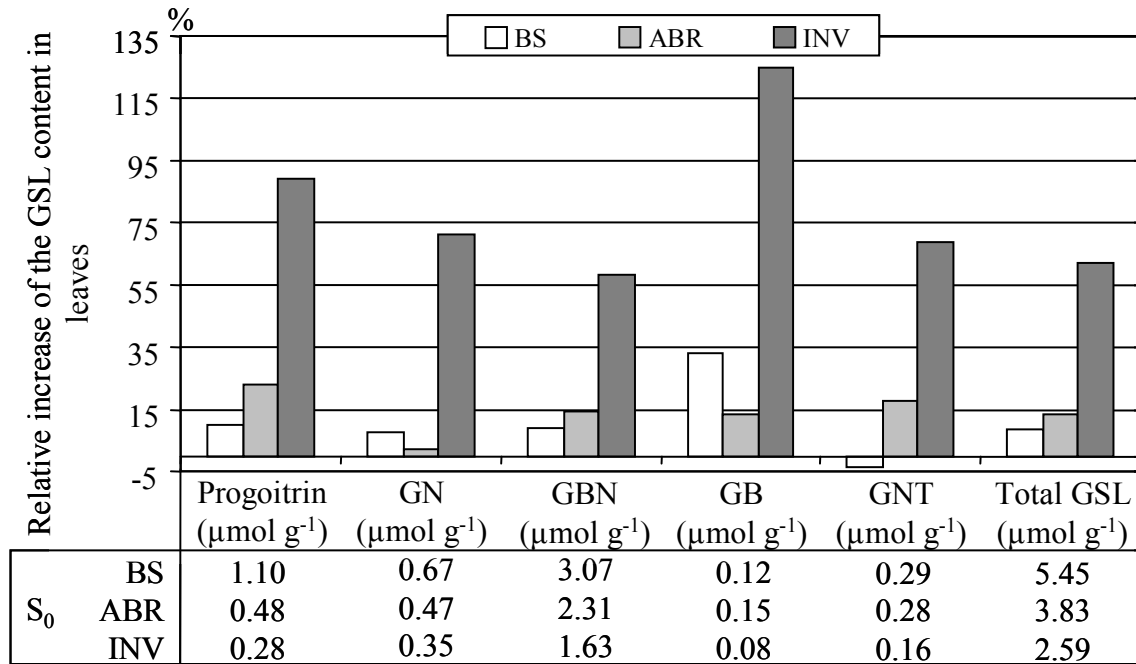


Figure 4.4: Relative increase of the individual and total glucosinolate (GSL) content in young leaves of winter oilseed rape in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (GN – gluconapin; GBN – glucobrassicinapin; GB – glucobrassicin; GNT – gluconasturtiin; relative increase in relation to control plots).

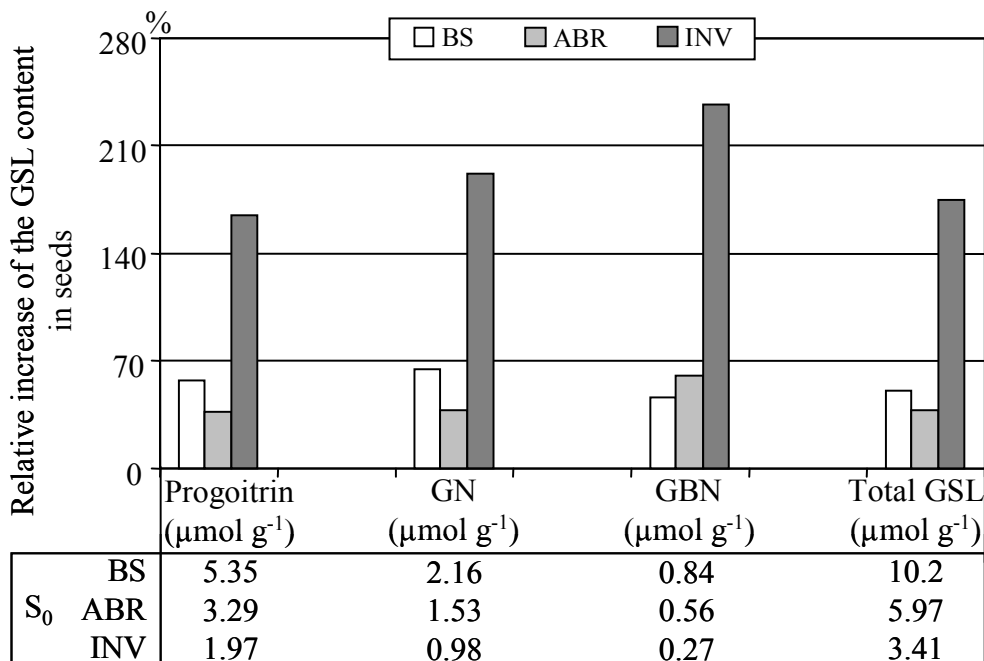


Figure 4.5: Relative increase of the alkenyl and total glucosinolate (GSL) content in seeds of winter oilseed rape in dependence on S fertilisation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002) (GN – gluconapin; GBN – glucobrassicinapin; relative increase in relation to control plots).

The total GSL content in leaves increased by 9%, 14% and 62% in Braunschweig, Aberdeen and Inverness, respectively (Figure 4.4), whereas for seeds the corresponding values were 51% in Braunschweig, 38% in Aberdeen and 175% in Inverness (Figure 4.5). Therefore, the strongly S deficient site in Inverness showed the highest increase in the content of total GSLs. A similar trend was also observed in the case of individual GSLs (Figure 4.4 and 4.5), and this could be again attributed to the phenomenon that the response to the S supply is stronger on S-deficient sites.

Glucosinolates have been reported to have biocidal properties, which fulfil the function of passive and/or active protective mechanism (Mithen et al., 1987; Schnug and Ceynowa, 1990; Doughty et al., 1991; Zúkalová and Vašák, 2002). Previous experiments indicated that in the infected plant tissues an increase in the concentration of indole and aromatic GSLs occurs (Koritsas et al., 1989; Doughty et al., 1991; Giamoustaris and Mithen, 1995), whereas the concentration of alkenyl GSLs declines (Doughty et al., 1991). However, the majority of work on the GSL induction has involved greenhouse trials and it is not clear to what extent the GSL induction occurs or can be detected in field grown plants. Field grown oilseed rape plants are constantly attacked by pests and pathogens and thus, they may be in a continued state of “induction”. While the induction of GSLs needs to be considered in studies investigating the significance of GSLs in plant defences, the sampling strategy adopted in the present study has prevented to note if the GSL induction take place or not. Regarding the constitutive defence mechanisms, glucobrassicinapin was shown to prevent the growth of the fungus *L. maculans* (Peterka and Schlosser, 1989), while progoitrin showed no effect or a small effect on the fungal growth (Mithen et al., 1987; Peterka and Schlosser, 1989). The relatively high proportion of glucobrassicinapin in the leaf tissue at the start of stem elongation (see Figure 3.4) together with the antifungal properties of this GSL might suggest a potential mean of increasing the resistance of plants to diseases. The significance of the GSL levels for the defence mechanisms of oilseed rape against *L. maculans* is discussed in chapter 4.5.

There have been conflicting reports on the effect of the N supply on the GSL contents (Herrmann, 1976; Booth et al., 1991). The present study indicated that the total GSL content in leaves was decreased by N application, while that from seeds was increased (see Figure 3.8 and 3.9). The way in which N effects the total GSL content is however dependent upon the nutritional N and S status of the crop at the beginning of the fertiliser trial (Schnug, 1991).

Schnug and Haneklaus (2000) reported that the significance of the initial N and S supply for the GSL content is probably due to physiological or root-morphological reasons.

The cultivars *Bristol* and *Lipton* were reported to have different susceptibilities to fungal pathogens. Part of the work reported here was to investigate if these differences are also reflected in the content of S metabolites. Differences could be confined to the parameters cysteine and progoitrin. For the cultivar *Lipton* a lower cysteine and a higher progoitrin content was found compared to the cultivar *Bristol* (see Table 3.8 and 3.9). Cysteine has been reported to be related to the resistance of plants against pathogens (Vidhyasekaran, 2000), while in the case of progoitrin contradictory reports on its biocidal properties exist (Mithen et al., 1987; Peterka and Schlosser, 1989). Although there is a faint possibility that the differences in the cysteine and progoitrin content between cultivars might be related to their different susceptibilities against fungal pathogens, both cultivars showed similar degree of infections by *P. brassicae* (see chapter 3.3) and differed in their susceptibility to *L. maculans* (see chapter 3.5).

The allocation of S into different S-containing compounds in young leaves under conditions of S deficiency and sufficient S supply is shown in Figure 4.6 (season 2001/2002). The two major pools of S in vegetative tissues were protein-S and $\text{SO}_4\text{-S}$, accounting for about 80% of the total S (Figure 4.6). Glutathione, which is undoubtedly essential for plant metabolism, accounted for approximately 15% of the total S in control plots and about 11% in S fertilised plots (Figure 4.6). Although GSLs are supposed to play a role as a S storage source (Schnug and Haneklaus, 1993a), they represented only a small fraction of the leaf S, accounting for about 3% of the total S in control and approximately 2% in S fertilised plots (Figure 4.6). These results are in agreement with the findings of Blake-Kalff et al. (1998), who reported that GSLs account for less than 5% of the total S in vegetative tissues. Cysteine was the smallest S fraction, accounting for less than 1% of the total S (Figure 4.6). On the low S sites in Scotland, relatively more S taken up by the plant is incorporated into organic compounds (Figure 4.6). These findings are in line with the investigations of Mengel (1991), showing that with increasing the S supply and absorption, the ratio organic S : inorganic S in plants decreases.

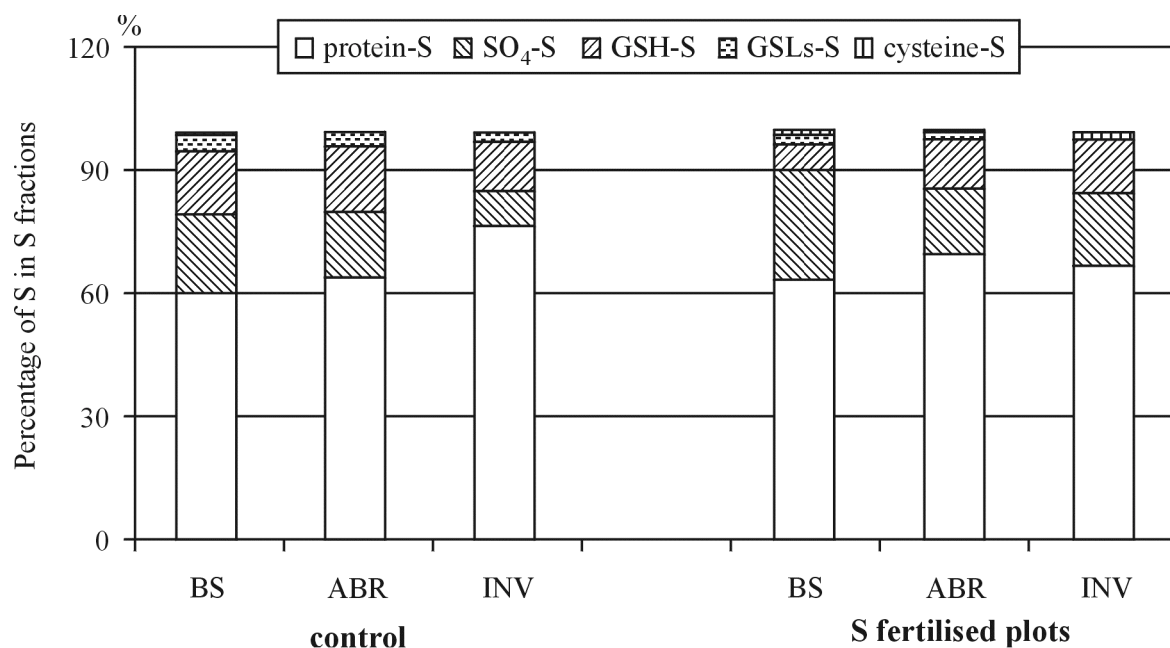


Figure 4.6: Distribution of S in young leaves of winter oilseed rape at the start of stem elongation in Braunschweig (BS), Aberdeen (ABR) and Inverness (INV) (season 2001/2002).

The results from the bivariate analysis, investigating the relationships between individual S fractions, generally revealed close correlations in both leaves and seeds (see Figure 3.10 to 3.13). Nevertheless, even if correlations between different S fractions are to be expected, they were not found in all seasons or they were poor. In the case of field experiments, weak but significant correlations are of high relevance to describe and understand relationships between different metabolites and metabolic processes because under natural conditions a great number of influencing factors are of significance. There were several diseases, insects, grazing animals like roe deer, and climatic conditions, which were influencing the crop growth and development and limited the S utilisation.

The correlation analysis between leaf and seed S fractions revealed a close relationship between the total GSL content in seeds and the total S content in young leaves. Schnug (1989) also reported about the dependency of the GSL content in seeds on the total S content in the vegetative tissue. The author concluded that on an average the variability of the S supply explains 50% of the variability of the GSL content in seeds at harvest.

The present findings underline the importance of an optimised S nutrition, varietal type and environmental conditions for the content of individual S metabolites in winter oilseed rape tissues. However, in the future it is very important to attempt to establish the optimum level of S

fertilisation require to fully satisfy the physiological demand of oilseed rape together with the S doses necessary for inducing S dependent resistance mechanisms in plants. The timing of S applications might be also essential for combating pests and diseases and therefore, fertiliser strategies should be developed in order to enhance *SIR* under field conditions.

4.3 Significance of the sulphur nutrition for crop resistance against fungal pathogens

Earlier reports revealed that the S nutrition plays a key role in promoting natural plant resistance mechanisms against pests and diseases (Schnug et al., 1995a; Bourbos et al., 2000; Klikocka et al., 2004). The present results showed that the application of S reduced the disease incidence and severity of the biotrophic fungus *P. parasitica*. Contrary, no effect of S fertilisation on *P. brassicae* and *L. maculans* infections could be demonstrated (see Figure 3.17 and 3.18). However, high levels of cysteine and γ -glutamyl-cysteine have proved to be related with low levels of *P. brassicae* infections (see Figure 3.19). This indicates that the stimulation of plant S metabolism by S fertilisation might provide efficient tools to induce resistance to pathogens.

The pathogens *P. brassicae* and *L. maculans* are hemi-biotrophic fungi. *Pyrenopeziza brassicae* has first a biotrophic phase after infection followed by a necrotrophic phase, whereas *L. maculans* has a necrotrophic phase on leaves followed by a systemic biotrophic phase in petioles (Ashby, 1997). The present findings, indicating that the disease incidence and severity of *P. brassicae* were not affected by S fertilisation, are contradictory with the investigation of Schnug et al. (1995a), who found that S reduced infections by *P. brassicae* in oilseed rape crop in Scotland. The discrepancy between these two experiments cannot be attributed to differences in the severity of epidemics between cultivars, between regions and between years. The cultivar *Bristol* was used in both experiments and both field trials were conducted in regions with low atmospheric S depositions in Scotland. The severity of epidemics was also not changed from the first experiment of Schnug et al. (1995a), where a value for disease incidence (cultivar *Bristol*) of 63-100% plants infected by *P. brassicae* at the beginning of stem elongation was reported (Sutherland, 2001). The lack of *SIR* towards *P. brassicae* observed in the present study might be a consequence of the simultaneous presence of multiple pathogen infections in oilseed rape crop or differences in the plant defence strategy.

Previous reports have shown that plants are able to distinguish between different types of biotic challenges and to activate one or more signal transduction pathways, which trigger finally the synthesis of different sets of chemicals or gene products in response to each type of challenge (Stout and Bostock, 2000). The signal transduction pathways leading to the activation of defence

reactions are only poorly understood. The majority of recent work is focused on two signal transduction pathways, one mediated in part by salicylic acid (SA), the other mediated in part by the octadecanoid pathway, of which jasmonic acid (JA) is a component. The SA pathway appeared to be associated with the activation of plant defence responses mainly to biotrophic pathogen infections (Hammond-Kosack and Parker, 2003), while the JA pathway is reported to be involved in the responses of plants mainly to necrotrophic pathogen attacks (Hammond-Kosack and Parker, 2003; Oliver and Ipcho, 2004) and to wounding and feeding by chewing insects (Stout and Bostock, 2000). The specificity of induced responses was demonstrated in the recent research with *Arabidopsis thaliana* (Thomma et al., 2002). The SA pathway was required for the full resistance against the biotrophic fungal pathogen *P. parasitica*, but not for the resistance against the necrotrophic pathogens *A. brassicicola* and *B. cinerea*. On the other hand, jasmonate signalling was shown to be required for the defence against *B. cinerea*, but not against *P. parasitica* (Thomma et al., 2002).

Increases in SA are associated with the expression of a number of PR proteins and of broad-spectrum resistance to pathogens (SAR) (Stout and Bostock, 2000). By applying SA to *Brassica* plants and tobacco leaves increased levels of certain GSLs (Doughty et al., 1995) and of the foliar GSH content (Fodor et al., 1997) were also reported. The SA synthesis, a CoASH requiring in the β -oxidation pathway (Ryals et al., 1996) with cysteine being one of the precursors of CoASH synthesis (Luckner, 1990), is somehow linked to the S metabolism of plants. On the other hand, increases in JA are associated with increases in the level of several compounds, including proteinase inhibitors and polyphenol oxidases (Stout and Bostock, 2000). JA increased also the transcript abundance of the enzymes involved in GSH synthesis without increasing the GSH content in *Arabidopsis* (Xiang and Oliver, 1998). Inhibition of the JA pathway by SA is the best documented and best understood antagonistic interaction. This occurs because SA blocks the pathway that culminates in the synthesis of JA and the transcription of genes coding for defences (Doares et al., 1995). Based on these accounts, then perhaps biotrophs that stimulate the SA pathway are indirectly depressing plant responses that would be most effective against necrotrophs. There are some studies to test this and one example is the relation between *B. graminis* and *M. grisea* resistance in barley (Jarosch et al., 2003). The authors showed that mildew resistance conferred by the *mlo* gene was accompanied by enhanced susceptibility to the necrotroph *M. grisea*. A similar antagonistic resistance relationship occurred between the biotroph *Puccinia coronata* and the necrotroph *Cochliobolus victoriae* in oats (Wolpert et al., 1994). However, most of the experiments reporting the negative cross-talk between the SA and JA pathway were performed in laboratory conditions and in most cases

involved the exogenous application of the hormones (SA or JA) and assays of the resistance to a single pathogen that activates either pathway exclusively. The role of these interactions in the resistance of plants in natural environments has not been thoroughly tested. Under field conditions, the plant may be challenged by numerous pathogens simultaneously and it has to activate a response that maximises fitness in each condition. Resistance responses are complex traits that involve large changes in metabolism and may impose major energetic costs to the plant. Thus, a trade-off may be established with the development of certain defences resulting in the deactivation of others.

In the present study, winter oilseed rape plants were simultaneously infected by three fungal pathogens, namely *P. brassicae*, *L. maculans* and *P. parasitica*. Because of these stress combinations, the plant fitness might have been adversely affected. The enhancement of the plant vigour by S fertilisation resulted in increased resistance against *P. parasitica*. SA has been reported to be linked to the response of *Arabidopsis* against infections by *Peronospora* (Cao et al., 1998; Thomma et al., 2002). This indicates that the SA pathway might have been also activated here in response to infections of winter oilseed rape by *P. parasitica*. Therefore, defence resistance compounds against this fungus might have been generated. Many of these defences are supposed to be S-containing compounds, preformed (e.g. GSLs) or induced following pathogen infections (e.g. phytoalexins) (Mohr and Schopfer, 1994; Sinha, 1995). When one considers the antagonistic interaction between signal transduction pathways, then the stimulation of the SA pathway might suppress the JA pathway and subsequently the induction of the defence mechanisms effective against *P. brassicae*. However, to develop a comprehensive picture of the pathways and responses triggered when a plant is challenged by multiple pathogens and/or stress, several milestones will have to be reached. The recognition of these pathways may help to explain the idiosyncratic pattern of specificity and cross-resistance observed among different pathogens, and the manipulation of these mechanisms may contribute to disease control. It might be possible that an adequate and regular S nutrition to plants might be essential to ensure the reserves for the production of elevated levels of S-containing metabolites and thus, plants will be better able to cope with fungal infections during the whole growth period.

4.4 Influence of infections by *Pyrenopeziza brassicae* on metabolite concentrations and enzymatic activities

The seasonal and regional variability observed in the present work regarding the infection of winter oilseed rape by *P. brassicae* most likely resulted from differences in the environmental factors (see chapter 2.1 and Appendix: Table A.1 to A.3) and in the distribution and the level of initial inoculum. Temperature and rainfall are of particular importance for spore liberation and dispersal of *P. brassicae* (Figuerola et al., 1995a and 1995b; Gilles et al., 2000). Rainfall was high during the second experimental season in Braunschweig (see chapter 2.1 and Appendix: Table A.1), and this might have encouraged the pathogen movement by water in the field and probably have promoted infections of oilseed rape by *P. brassicae* in this season. At the Aberdeen site, the infection pressure for *P. brassicae* is generally very high. The presence of the initial inoculum together with the high levels of precipitation falling during the experimentation (see chapter 2.1 and Appendix: Table A.2) might have stimulated the development of severe *P. brassicae* epidemics in winter oilseed rape crop on this site.

In the present research work, no differences were found between cultivars with respect to the disease incidence and severity of *P. brassicae* (see chapter 3.3), and this contrast with their apparently different susceptibility to this fungus (Gladders et al., 1998; HGCA Recommended List WOSR 2003). The cultivar *Bristol* was excluded for a long time from trials because of its high-recorded susceptibility to *P. brassicae* and other cultivars rated as resistant were introduced (e.g. *Lipton*). It might be possible that different pathotypes of *P. brassicae* have been formed during this period in order to specialize on these new cultivars. Therefore, the cultivar *Lipton* rated before as resistant might become susceptible to the new pathotypes. There are some evidences that sources of resistance to *P. brassicae* in new cultivars are being overwhelmed in a few seasons by changes in the pathogen population (Karolewski et al., 2004).

Among the S-containing metabolites putatively involved in *SIR*, cysteine, GSH and the enzymes supposedly involved in the H₂S release have received attention in the present study with respect to their role in the defence mechanisms of oilseed rape against *P. brassicae*. The focus here was to investigate if there is a relationship between the S nutritional status, the cysteine and GSH content, the potential of plants to release H₂S and fungal infections. The discussion of the results will be focused specifically on two aspects that are important for the understanding of *SIR* in plant defence mechanisms. First, the impact of *P. brassicae* infections on S metabolite concentrations and enzymatic activities will be assessed, followed by a

discussion on the potential of the cysteine and GSH pool towards other components of the cellular defence system.

Plants respond to pathogens by transient increases in the production of reactive oxygen species (ROS) and therefore, they have evolved antioxidant defence systems to keep ROS under control. GSH is considered part of the antioxidative systems of plants, but till now no clear picture has emerged about the exact roles of GSH in infected plants (Tausz, 2001; Tausz et al., 2003). Regarding the GSH levels, most of the previous research has shown that in infected resistant plants there is an increase in the GSH content at the site of infection at the time when pathogen development is being arrested (Vanacker et al., 1998; May et al., 1998; Gullner and Kömives, 2001; Williams et al., 2002). The increase of the GSH levels in alfalfa and bean cells after treatment with fungal elicitors has been demonstrated by Edwards et al. (1991). Markedly increased GSH levels were also found in leaves and apoplasts of resistant barley after inoculation with the biotrophic fungus *Blumeria graminis* (Vanacker et al., 1998). The accumulation of elemental S and transient increases in the concentration of sulphate, GSH and cysteine were observed in tomato plants inoculated by the fungal vascular pathogen *V. dahliae* as well (Williams et al., 2002). Based on these findings, it has been assumed that the increase of the GSH content at the site of infection might operate through increased resistance of plants against fungal pathogens (Gullner and Kömives, 2001). However, in these studies, GSH levels were measured immediately after infection or with a delay of some hours/days when the pathogen is supposed to be in the first stages of development. The question arises whether plants respond similar in the case of more severe infections. There are some previous evidences that changes of the GSH content in response to drought, which is an abiotic factor, depend on the extent and intensity of the drought stress (Smirnoff, 1993). With more severe stress a degradation of the total GSH concentration was reported, which suggested a weakening of the defence system as a symptom of initial cell disruption (Smirnoff, 1993; Tausz, 2001). When one considers that GSH appears to be a universal response in plants faced by environmental stress and pathogen attack (Foyer and Rennenberg, 2000), it might be assumed that plant responses to fungal pathogens depend also on the extent and severity of the biotic stress.

In Figure 4.7, the relative changes of the cysteine and GSH content in the leaf tissue of winter oilseed rape due to infections caused by *P. brassicae* and in dependence on the S supply are shown. Marked differences were found with respect to the investigated parameters between experimental sites and seasons. This discrepancy would seem to stem from differences observed in the disease severity of *P. brassicae*.

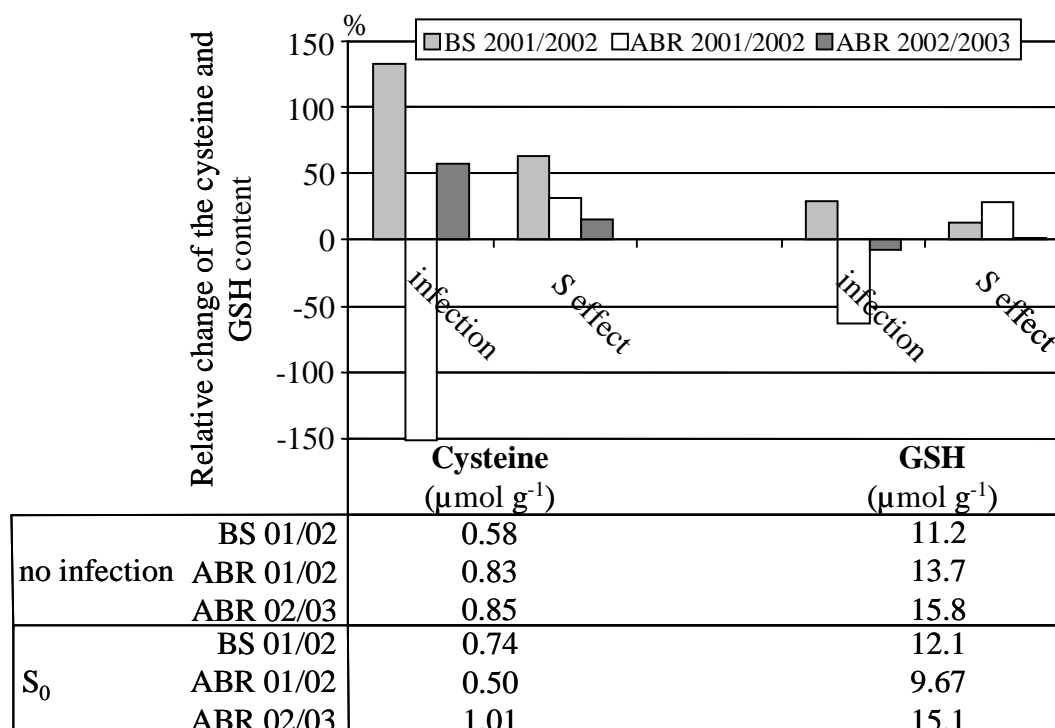


Figure 4.7: Relative changes of the cysteine and glutathione (GSH) content in leaf discs of winter oilseed rape at the start of stem elongation in relation to *P. brassicae* infections and in dependence on S fertilisation in Braunschweig (BS) and Aberdeen (ABR) (relative changes in relation to non-infected leaf discs/control plots).

In Braunschweig, where slight infections of winter oilseed rape leaves by *P. brassicae* were present at the time of sampling, an increase of the cysteine content by more than 130% and of the GSH content by about 30% was observed when plant material was visually infected by this fungus (Figure 4.7). Additionally, the increase in the level of these two parameters caused by the infection was even stronger than that caused by S fertilisation (Figure 4.7). There might be two possible explanations for the increase of the cysteine and GSH content as a result of *P. brassicae* infections. Firstly, the rate of cysteine synthesis may be tailored to the demand of cysteine in the synthesis of GSH. GSH may play itself a role in the plant defence system or may be further on metabolised to other compounds (e.g. phytoalexins), promoting in this way resistance. For instance, increased GSH levels in the plant tissue caused by the pathogen attack would be linked with increased ascorbic acid concentrations, which regulate the activity of the enzyme myrosinase and thus, the cleavage of GSLs and the release of toxic isothiocyanates (Schnug and Haneklaus, 1993a). Secondly, the increase of the cysteine content may be required for the synthesis of other metabolites which are derived from cysteine and which are necessary for the defence mechanisms of plants (e.g. H_2S ; see later on). These findings provide further evidences

that a rapid accumulation of cysteine and GSH in response to pathogen attack might be a response to fungal infections and thus, it might substantially contribute to plant resistance.

On the other hand, in Aberdeen, high infection rates of *P. brassicae* at the time of sampling (see chapter 3.4 and Appendix: Table A.19 and A.21) had made almost impossible the collection of leaf disc samples from infected and non-infected leaves, respectively. Therefore, visually non-infected leaf discs might have not been free of infection even if no symptoms were detected. Moreover, in the second season, there was a competition between different fungi on the same plant (see chapter 3.3 and 3.4 and Appendix: Table A.19). In the second season, a decrease of the cysteine and GSH content by about 150% and 60%, respectively was found in infected leaf discs (Figure 4.7). Nevertheless, the decrease in the content of these two metabolites does not necessarily indicate a higher susceptibility of oilseed rape crop to *P. brassicae*. There might be two possible explanations for this situation. First, the drastic decrease observed in the content of cysteine and GSH might be due to a change in the number of living cells. It cannot be excluded that in the case of strong infected leaves a great amount of cells are already dead. Consequently, even when in the living cells the GSH level remains constant or increases, in the overall basis (leaf discs) the content might decrease. Nevertheless, leaf discs collected showed no visible necrosis and thus, there is a remote possibility that the presence of dead cells is the cause of the decrease. Second, it might be possible that a critical infection threshold exists above which changes in plant responses arise. Whether in the case of initial infections an increase of the cysteine and GSH content was observed (see above), with more severe stress a decrease of these two pools might occur. This decrease might suggest that the rate of cysteine and GSH synthesis has been directed to the demand of other S related defences (e.g. phytoalexins, GSLs), contributing in this way to resistance.

In the third season in Aberdeen, when only infections of winter oilseed rape leaves by *P. brassicae* were observed at the time of sampling and the disease severity was lower compared to the second season (see Appendix: Table A.19 and A.21), an increase of the cysteine content by about 60% and a decrease of the GSH level by 7% were found (Figure 4.7). Even if the GSH content decreased slightly when compared to non-infected leaf discs, an increased GSH content was found in the infected leaf discs that showed high infections by *P. brassicae* (see Figure 3.23). This might indicate that high levels of GSH were required in these tissues in order to cope with enhanced levels of ROS. These findings suggest again that high levels of cysteine and GSH present in infected tissues might have some protective function.

When compared to whole leaf samples, the cysteine and GSH levels found in leaf discs were quite small (see Table 3.6, 3.12 and 3.13). It might be possible that by sampling leaf discs marked alterations of the GSH pool occurred, alterations that does not take place by sampling whole leaves. However, the metabolic changes in the S metabolite concentrations triggered by the presence of a fungal infection are supposed to be better revealed at the site of infection. Therefore, whole leaf samples might not be of confidence in identifying the defence S compounds involved in plant resistance. Leaf disc samples might reveal better the responses of plants to biotic challenges and thus, they should be taken into account when investigating chemical defences.

The involvement of H₂S in the resistance of crops against fungal pathogens has been proposed by Schnug et al. (1995a), but from this report the significance of toxic H₂S emissions for natural plant resistance mechanisms is still not proved. Rennenberg (1983) reported that the path of H₂S biosynthesis in response to sulphate seems to be desulphhydration of L-cysteine. It has been also found before that oilseed rape varieties contain different levels of the enzymes responsible for the degradation of cysteine to H₂S (Burandt et al., 2001). Therefore, the rate of H₂S synthesis might be influenced either by the supply of cysteine, by the modulation of the enzyme activities (LCD and OAS-TL), or by both. In the present research work, the influence of infections by *P. brassicae* on the cysteine content (see above) as well as on the LCD and OAS-TL activity has been studied.

In Figure 4.8, the relative changes of the LCD and OAS-TL activity in relation to *P. brassicae* infections and in dependence on S fertilisation are shown. Since cysteine is the substrate for LCD, an optimised S nutrition will increase the substrate availability for the enzyme. But while results clearly showed an increase of the LCD activity when the plant tissue was infected by *P. brassicae*, S fertilisation led to a decrease (Figure 4.8). The increase of the LCD activity by about 50% in the infected tissue is probably associated with the release of fungitoxic H₂S, promoting in this way resistance to *P. brassicae*. It can be speculated that a higher LCD activity will result in a higher H₂S release.

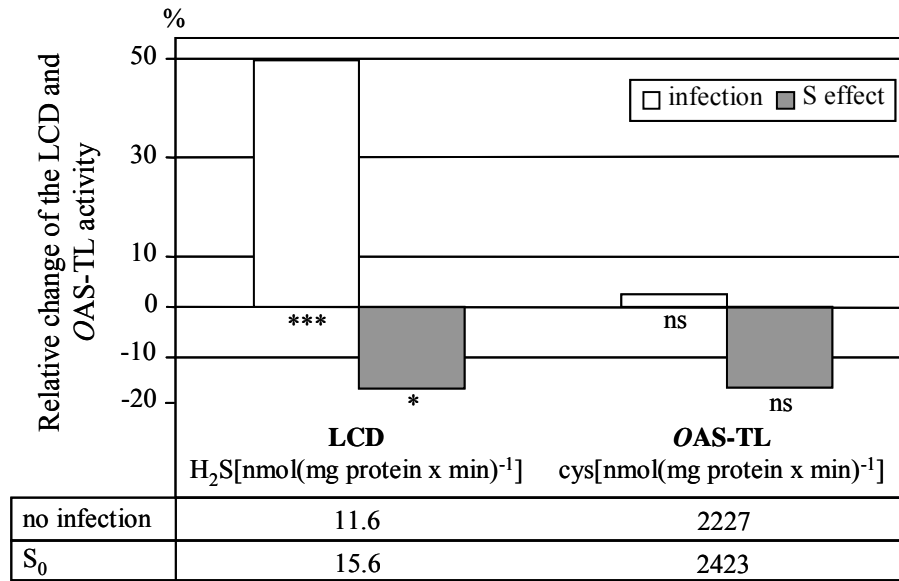


Figure 4.8: Relative changes of the L-cysteine desulphhydrase (LCD) and *O*-acetylserine(thiol)lyase (*OAS-TL*) activity in leaf discs of winter oilseed rape at the start of stem elongation in relation to *P. brassicae* infections and in dependence on S fertilisation (Braunschweig, 2001/2002) (relative changes in relation to non-infected leaf discs/control plots).

On the other hand, the *OAS-TL* activity was neither influenced by the S nutrition, nor by pathogenesis (Figure 4.8). These results suggest that *OAS-TL* is not actively increasing the H₂S release with infection, but might participate in increasing the H₂S release in a passive way (Bloem et al., 2004). The minor role of *OAS-TL* in the H₂S emission has been also revealed in studies of Burandt et al. (2001). The authors showed that in a molar ratio the enzyme formed about 25 times more cysteine than H₂S per mg protein during the same incubation time. It was demonstrated in yeast as well, that the simple hypothesis that variation in the *OAS-TL* activity is correlated with the H₂S production and release is not supported by any data (Spiropoulos and Bisson, 2000).

The fact that *OAS-TL* was not significantly up-regulated while the product of reaction increased strongly (i.e. cysteine; see above) probably shows that the enzyme activity was high enough to allow a fast turnover from sulphate to cysteine in the plant. *OAS-TL* is most likely regulated by the N assimilatory pathway, because the *OAS-TL* activity significantly increased by N fertilisation (see Table 3.14). An increase in the activity of *OAS-TL* and APS reductase under N sufficient conditions was also reported in *Lemma minor* L. and cultured tobacco cells (Reuveny et al., 1980; Smith, 1980; Brunold and Suter, 1984; Koprivova et al., 2000). The activity of LCD also increased with N fertilisation (see Table 3.14) probably to prevent the plant

from a too high and toxic cysteine pool. Several studies established regulatory interactions between assimilatory sulphate and nitrate reduction in plants (Brunold, 1993; Kopriva and Koprivova, 2003). The two assimilatory pathways are interrelated: changes in the N supply affect the S demand of plants and vice versa (Schnug and Haneklaus, 2000; Koprivova et al., 2000). Altogether, the sulphate reduction seems to be regulated by the N nutrition on transcriptional level and *OAS* plays a major role in this regulation (Koprivova et al., 2000). In the presence of excess sulphate, *OAS* seems to be limiting for cysteine synthesis (Rennenberg, 1983). In comparison, *OAS* accumulates during S starvation and thus, it may act as a signal of the S status (Kim et al., 1999). *OAS* acts most probably as a transcriptional regulator since a higher *OAS* content strongly increased mRNA levels of APS, sulphite reductase, chloroplastic *OAS*-TL, and cytosolic SAT (Kopriva and Koprivova, 2003).

The fact that the activity of *OAS*-TL and LCD was higher in S deficient plants (see Figure 4.8) indicates that under conditions of S deficiency the S metabolism is activated and the participating enzymes are up-regulated. There are two possible explanations: S deficient plants were more susceptible to fungal diseases and therefore they increased the metabolic pathways, which are involved in plant protection. The second explanation could be that S deficient plants had already a stronger fungal infection and the mechanisms of *SIR* were activated. Therefore, the release of H₂S can be a mechanism of protection to prevent a fungal attack or to answer to a fungal attack or probably both mechanisms are working at the same time (Bloem et al., 2004).

The results from the correlation analysis revealed a strong positive relationship between the cysteine, γ -glutamyl-cysteine and GSH content (see Figure 3.21). This implies that factors that stimulate GSH synthesis (e.g. fungal pathogen) will promote an increasing demand upon S assimilation into cysteine and subsequently γ -glutamyl-cysteine. These findings are in line with earlier studies that strongly support the view that the cysteine availability in plants plays an important role in determining cellular GSH concentrations through kinetic restriction of the reaction catalysed by γ -ECS (Brunold and Rennenberg, 1997; Noctor et al., 1997). On the other hand, the cysteine, γ -glutamyl-cysteine and GSH content showed no correlations with the LCD and *OAS*-TL activity (see Figure 3.21). When the correlation analysis was carried out without differentiation between infected and non-infected leaf discs, strong positive correlations were found between the LCD activity with the cysteine and GSH content (Bloem et al., 2004). Both enzymes, LCD and *OAS*-TL were positively correlated with each other in both infected and non-infected leaf discs as well as with the total N content (see Figure 3.21 and 3.22). The positive relationship between the LCD and *OAS*-TL activity indicates that also the activity of

OAS-TL is increasing after fungal infection, but not as a direct result of infection rather than as a reaction to the activity of *LCD*, which is consuming cysteine the product of *OAS-TL* reaction. Burandt et al. (2001) found an inverse relationship between these two enzymes for different genotypes of oilseed rape ($r = -0.675$; $p < 0.05$). These crops received the same rate of S fertilisation, but differed in their susceptibility to different fungal diseases. This stresses the significance of genetic differences and putatively involved modifications of the S metabolism. However, no differences between *Bristol* and *Lipton* with respect to the *LCD* and *OAS-TL* activity were found in the present study. This might indicate that these two cultivars have the same potential to release H_2S in response to *P. brassicae* infections. The correlation analysis also revealed weak negative correlations between the enzyme activities and the total S content and this is in line with the investigations of Burandt et al. (2001). As outlined in chapter 4.2, weak correlations are of high significance under field conditions because a wide variety of factors might influence the crop growth and development.

The present results revealed that the metabolic changes triggered in the plant tissues by the presence of fungal infections might be dependent on the developmental stage of the pathogen. Cysteine and GSH might be potential response metabolites to initial fungal infections, whereas in the case of severe infections a diminution of these two pools might occur. The results also revealed that *OAS-TL* is not actively increasing the H_2S release with infection. On the other hand, *LCD*, which activity is directly induced by infections of winter oilseed rape leaves by *P. brassicae*, seems to be the enzyme involved in the H_2S release. These findings are a strong evidence that the evolution of H_2S on the leaf surface may significantly contribute to develop natural plant protection measures. Therefore, the emission of H_2S from plants may be an important part of *SIR*.

4.5 Application of geostatistical analysis for the identification and interpretation of infections caused by Leptosphaeria maculans

SIR runs parallel to other defence mechanisms, which are activated after fungal attack, so that a clear differentiation is difficult, particularly under field conditions (see chapter 4.3). In the present research work, geostatistics were applied in order to capture the influence of the S nutritional status on the S-containing metabolites against the background of other factors that had an impact on them and to understand the factors governing the pathogenesis in dependence on the S supply under natural conditions. Geostatistics have been applied before in epidemiological studies for the analysis of the spatial variation of plant diseases on plot and field scales (Chellemi

et al., 1988; Larkin et al., 1995; Wu et al., 2001; Morgan et al., 2002). The utility of geostatistics as a method to measure differences between patterns of initial inoculum in soil and patterns of infected plants was demonstrated in studies of Chellemi et al. (1988). Spatial patterns for soil population levels of *Phytophthora capsici* and soil water content were characterised using geostatistical techniques in commercial bell pepper fields by Larkin et al. (1995). Using the same approach, Morgan et al. (2002), Barclay et al. (1973) and MacKenzie (1981) found that controlled N fertiliser management could reduce the infection rate and severity of early blight (*Alternaria solani*) in potatoes in small plot experiments. Geostatistics were also applied in the investigations of Wu et al. (2001) to provide a systematic quantitative analysis of the distribution of lettuce downy mildew (*Bremia lactucae*) in the Salinas valley.

The risk of severe *L. maculans* epidemics has not been quantified before, and this research work is the first to our knowledge, which provides the probability distribution of *L. maculans* infections in oilseed rape crop. Another advantage of using geostatistics in this context is that mechanisms and interactions involved in *SIR* of plants to fungal pathogens became visible, while they remained concealed by employing standard statistical procedures.

The semi-variogram is an adequate tool to analyse the spatial autocorrelation in dependence on distance and direction (Isaaks and Srivastava, 1989). The results from the semi-variogram analysis revealed that no spatial autocorrelation existed for the parameters total S and total GSL content and that only a moderate spatial dependency was found for GSH and the probability of severe *L. maculans* infections (see Table 3.16). For this reason, variability was uncovered within single plots, which indicates that micro-environmental conditions influenced the host-pathogen relationship.

The high nugget variance in the semi-variogram for GSH (see Table 3.16) indicates a large point-to-point variation at short distances and suggests that shorter sampling distances will reveal more details.

The ranges for spatial correlations are related to the scale of the experiment and the type of the pathogen involved. In the present research work, the range was about 8 m for the parameters probability of severe infections by *L. maculans* and the GSH content (see Table 3.16). Chellemi et al. (1988), using small study plots (3.6 x 3.6 m grid), determined ranges of about 2 m for infections of pineapple by *Phytophthora nicotianae*. Farias et al. (2002) reported ranges of 8.5 m for *Rotylenchulus reniformis* populations in a cotton field. On a field scale with larger sampling distances, Munkvold et al. (1993) reported ranges of 15-25 m for *Eutypa dieback* disease of grapes, Lecoustre et al. (1989) found ranges of > 120 m for the whitefly-vectored African

cassava mosaic virus, and Webster and Boag (1992) determined a range of 60 m for soil contaminations by the nematodes *Globodera rostochiensis* and *Heterodera avenae* on potato fields.

The results from digital maps revealed distinct variations for the probability of strong *L. maculans* infections. The cultivar *Lipton* showed a higher susceptibility against this fungus compared to the cultivar *Bristol*. This finding agrees those of Gladders et al. (1998) and HGCA (2003). The maps also indicated that the resistance towards *L. maculans* was obviously related to the S status of the plant. In areas where the risk of severe infections was low a high total S and total GSL content was found (see Figure 3.25). Therefore, a sufficient S supply during plant growth as well as an adequate availability of plant available sulphate is presumably a prerequisite in order to promote resistance against fungal pathogens. So that, the required S rates may be higher than the physiological demand (Haneklaus et al., 2004). Regarding GSLs, their concentration in the leaf tissue of *Brassica* crops have been estimated to be sufficient to inhibit fungal growth (Greenhalgh and Mitchell, 1976; Mithen et al., 1987). However, there are contradictory reports on the phytopathological aspects of GSLs. Whereas Mithen et al. (1987), Peterka and Schloesser (1988), Koch (1989) and Schnug and Ceynowa (1990) have demonstrated a relationship between the GSL levels and the resistance of oilseed rape to the pathogen *L. maculans*, Giamoustaris and Mithen (1997) showed that increasing the total GSL content in the leaf tissue is not likely to lead to enhanced resistance to *L. maculans*. The results of the present experiment suggest that elevated levels of GSLs might contribute to the resistance of winter oilseed rape to *L. maculans*. Taking into account that glucobrassicinapin was the predominant GSL in the leaf tissue and consider that this GSL was reported to prevent the growth of *L. maculans* (Peterka and Schloesser, 1989), it can be speculated that the high levels of glucobrassicinapin might have been related to increased tolerance towards *L. maculans*. However, much more work is required to understand the interactions between pests and diseases and the individual GSLs.

The present results also indicated that both cultivars responded to *L. maculans* infections by an increased GSH synthesis, whereby the onset of synthesis seemed to be dependent on some threshold infection (see Figure 3.25). Therefore, further evidences are provided that the accumulation of GSH in response to pathogen attack is one response mechanism to fungal infections (see also chapter 4.4) (Vanacker et al., 2000; Gullner and Kömives, 2001).

The results from the geostatistical analysis revealed that the probability of severe *L. maculans* infections could be set in relation to plant physiological parameters and thus, it substantially contributed to the identification of metabolic processes that entail *SIR*. There are numerous diseases whose distribution can be analysed in this way and whose causes might be sought in the plant characteristics by comparing the respective spatial patterns. Therefore, the geostatistical analysis of plant, soil and disease data is a useful strategy for improving the studies on interactions between pests and diseases and individual S fractions in a given specie and cultivar. Moreover, these spatial correlation patterns might help us to develop control strategies under particular field conditions and also to improve sampling strategies at different scales in the future.

4.6 Verification of the SIR concept employing field experimental data from different sites

There are two undeniable factors that have been demonstrated by previous studies on the interactions between S and pathogens: resistance exists and resistant plants quickly react to pathogen ingress with a number of different S responses (e.g. cysteine, GSH, GSL, phytoalexins) (Haneklaus et al., 2002 and 2004). The putative defensive role of the S-containing compounds was supported by *in vitro* assays, which suggest that the response being studied can play some defensive role (Mithen et al., 1987; Bohlmann, 1999; Hammerschmidt and Nicholson, 2000). Subsequently, with the progress in the molecular era of plant biology, the specific role of the S metabolites in disease responses was evaluated using transgenic plants developed with genetic engineering techniques. The expression of these compounds in transgenic plant species has shown that the development of fungal pathogens can be significantly reduced (Gao et al., 2000). Additionally, using mutants that lack the ability to synthesise or accumulate each of these compounds, or manipulation of salicylic acid, ethylene and jasmonate levels have also provided some interesting results with regard to enhanced disease tolerance and susceptibility (Doughty et al., 1995; Thomma et al., 2002). All these studies are valuable because they demonstrate that the compound being studied is directly involved in defence, but they are not able to provide definitive proof that the S-containing metabolites act in the same way in the native host/pathogen interactions. Therefore, it was a need to evaluate plant defences to pathogens on field trials and appropriate agronomic practices to ensure that the S-containing compounds play a decisive role in pathogenesis. Due to productive collaboration between the Institute of Plant Nutrition and Soil Science, FAL, Braunschweig and the Scottish Agricultural College (SAC), Aberdeen, Scotland, a significant progress in understanding the mechanisms and processes causing *SIR* was achieved.

The main aim of the present research work, namely to identify the S-containing metabolites involved in *SIR* in dependence on the S supply under field conditions, was achieved by using either infection-directed sampling strategy or geostatistical analysis. Figure 4.9 illustrates the S metabolites and pathways putatively involved in the chain reactions of *SIR* in *Brassica* species according to Haneklaus et al. (2004). The present results showed that the cysteine, GSH and GSL content in the leaf tissue of oilseed rape is influenced by the S supply, and this indicates that a good S nutrition can enhance the capability of plants to cope with biotic stress. The changes found in the concentration of these metabolites together with the changes observed in enzymatic activities (LCD and OAS-TL) triggered by the presence of fungal infections are added to the figure (Figure 4.9). Two possibilities can be identified: initial infections and infections in the late developmental stage of the pathogen.

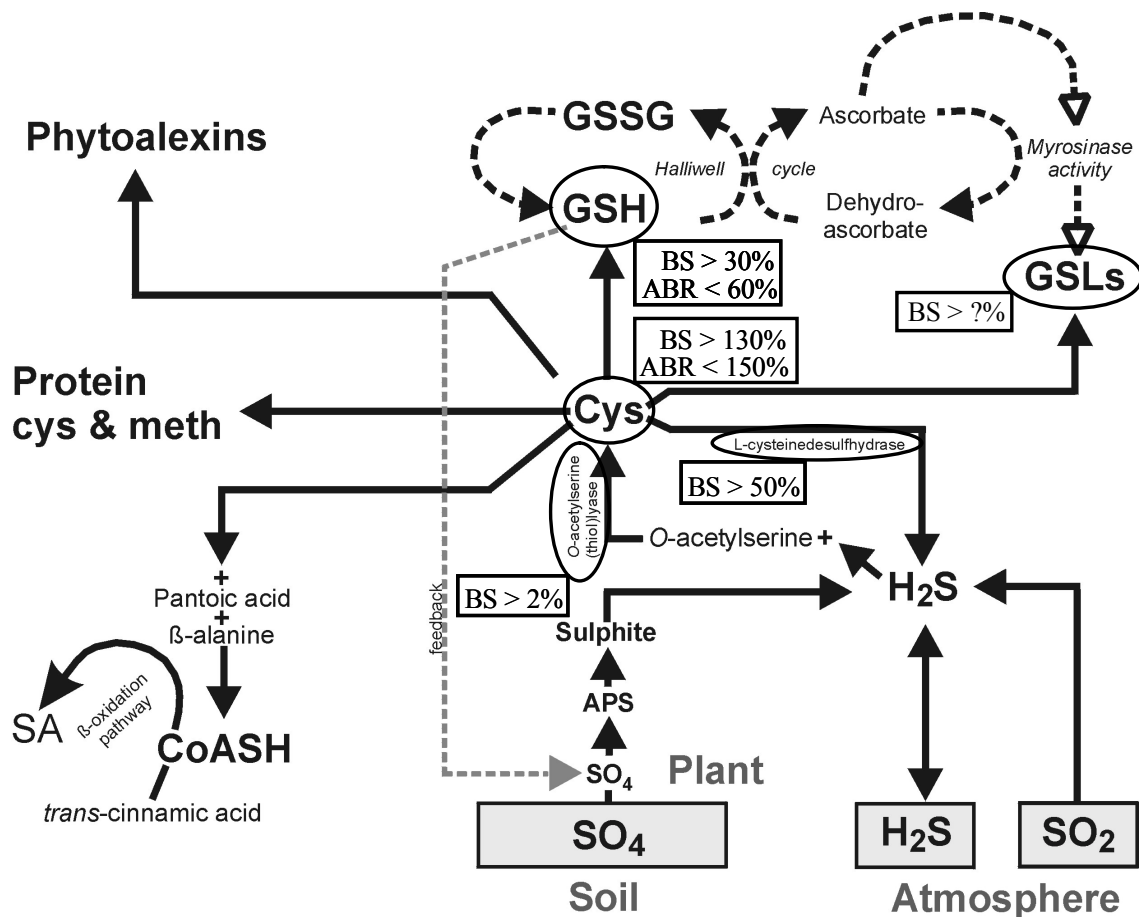


Figure 4.9: Sulphur metabolites and pathways putatively involved in chain reactions of *SIR* in *Brassica* species (according to Haneklaus et al., 2004) (BS – Braunschweig; ABR – Aberdeen; > or <: metabolic changes (increase/decrease) as a result of fungal infections).

When infections are in the initial stages, considered to be the case in Braunschweig, an increase of the cysteine and GSH content was noted at the site of pathogen attack (Figure 4.9). High levels of GSLs also proved to be related with reduced fungal infections. In this case, elevated levels of these metabolites may play significant roles in the plant defence system against fungal pathogens. Additionally, substantial evidences were provided that the evolution of H₂S on the leaf surface by the action of LCD is related to the crop fungal infection and therefore, it may also significantly contribute to develop natural plant protection measures (Figure 4.9). However, to yield a significant fungicidal effect on pathogens, the emissions of H₂S by plants need to be higher than a specific threshold supported by fungi. Up to now no information exists about fungitoxic H₂S concentrations on fungal spores and mycelia, so that it is possible that the H₂S emission of the plant acts as an inhibitor of spore germination and/or yields a fungitoxic effect on the vegetative phase of the fungi (Haneklaus et al., 2004). Work on this topic and measurements of gaseous S emissions from plants in dependence on fungal infections are currently underway at the Institute of Plant Nutrition and Soil Science, FAL, Braunschweig.

In the case of infections in the late developmental stage of the pathogen, as possible present in Aberdeen, decreased levels of cysteine and GSH at the site of infection might reflect either a biochemical damage of the tissue or a rapid turn into other S defence related compounds (Figure 4.9). For instance, the induction of phytoalexins around the infected area might provide efficient tools to combat fungal attacks.

The present results would support the fact that plant responses to biotic stress might depend on the extent and severity of infection. However, further work is required to determine the extent of differences under different stress intensities. The measurements of plant responses during the whole developmental stage of the pathogen should therefore provide insights into the similarities and differences between biochemical mechanisms of resistance to different stress conditions.

It has been also demonstrated here that the role of S in natural defences of plants depends not only on the behaviour of a particular pathogen and its developmental stage, but also on interactions between pathogens on the crop. Previous field investigations revealed that S has a repressive effect on different types of fungi (biotrophs or necrotrophs) (Schnug et al., 1995a; Bourbos et al., 2000; Klikocka et al., 2004), but this depends again on the timing, form and doses of S applications. For instance, fall S fertilisation is required for fungal pathogens that start infecting plants early in the fall (e.g. *P. brassicae*). When plants are simultaneously challenged by numerous pathogens and each pathogen has different life cycle, then S applications might effectively overcome some, but not all infections. The present investigations revealed that S was

effective against *P. parasitica*, but not towards *P. brassicae*. Therefore, S induced resistance to multiple pathogen infections might not be independently regulated. Under prolonged disease-conductive conditions and/or multiple pathogen interactions, a regular S fertilisation throughout the growing season, from seed to harvest, might provide significant advantages for disease management in comparison to one or two times S applications. Thus, a continuous S supply to plants will maintain the plant vigour and will ceaselessly stimulate the biochemical processes in the primary and secondary metabolism. Healthy plants will be therefore better able to withstand infections.

More evidences are therefore provided by the present field experiments that S may have a protective effect against fungal pathogens by the activation of plant S defences. However, plants with a higher cysteine, GSH or GSL content, or higher enzymatic activities, may or may not be resistant when the whole plant system is observed because the plant response is a concerted action of the whole metabolism (Hammerschmidt and Nicholson, 2000). The parameters studied here are only a few components of the plant defence system. To have a complete certitude of the effectiveness of *SIR* in plant defences attention has to be given to all putative S constituents. It is equally important to understand how these S-containing compounds may participate in the mechanisms by which plants deploy their defences and modify their growth and development in an effort to tune their sessile lifestyle to biotic constraints. It remains also to be demonstrated if *SIR* will fit into the natural and agronomical environments involving multiple species interactions and multiple trophic levels, and to which level the disease resistance is achieved by S fertilisation, and whether it is against a range of phytopathogens or only specific diseases. Therefore, in order to explore relationships between S metabolites and the resistance of plants to pests and diseases, further experiments with cultivars showing a high variability in their susceptibility against pathogens and a multivariate analysis of multiple S constituents of the plant defence system are required. Leaf disc sampling should be included because, as demonstrated here, these sampling strategies proved to better reveal the metabolic changes triggered in the plant S metabolism by the presence of a fungal infection. Geostatistics may also provide numerous research opportunities to investigate the plant pathogen population dynamics and discover interactions among plant pathogens and plant parameters.

Once the mechanisms and triggers causing *SIR* are completely understood, strategies can be developed to optimise *SIR* with view to the application form, the rate and timing of S fertilisation. Then, *SIR* can be successfully implemented in farmer's field and will contribute to a sustainable agricultural production that meets consumer demands.

5 Summary

The main objective of the present research work was to investigate the effect of the S nutrition on S-containing metabolites in relation to fungal infections. The research strategy was based on the hypothesis that adjusting the S nutrition of the growing crop, and thus stimulating the biochemical processes in the primary and secondary S metabolism, can reduce and/or slow down fungal infections. Apart from increasing the knowledge on the significance of S metabolites for plant resistance, the relevance of this work is given by the implementation of infection-directed sampling strategies and geostatistical analysis in order to achieve an advanced understanding of the interactions between pests and diseases and individual S fractions under field conditions.

The investigations were conducted on three sites which differed in climate, soil type and atmospheric S depositions: Braunschweig (E 10° 27', N 52° 18'), Aberdeen (W 2° 13', N 57° 12') and Inverness (W 4° 5', N 57° 53'). The S demand of oilseed rape was provided by the application of sulphate to the soil, avoiding therefore the potential direct fungicidal effect that may have been associated with foliar applied S. Among the putative S-containing defensive compounds, attention has been focussed on cysteine, glutathione (GSH), glucosinolates (GSLs) and enzymes involved in the H₂S release because of their dependence on the S nutritional status of the crop.

The main results of the work presented here were:

- 1) S fertilisation increased slightly (non-significantly) the seed yield on all three experimental sites, while N fertilisation and fungicide treatment had a significant effect on crop productivity.
- 2) S fertilisation had a significant positive effect on all S fractions investigated (e.g. total S, SO₄-S, cysteine, GSH, GSLs), indicating that with a better S supply the potential of *SIR* is increasing. Marked differences were observed between experimental sites and seasons, suggesting among others wide ranges of S availabilities. The highest responses to S applications were found on the low S site in Inverness.
- 3) High rates of N fertilisation increased the total N content in young leaves and the total S and total GSL content in seeds.

- 4) Differences between cultivars were confined to the parameters cysteine and progoitrin content as well as to the final seed yield. The cultivar *Lipton* contained less cysteine and more progoitrin and yielded more than the cultivar *Bristol*.
- 5) Disease assessments carried out in Scotland revealed that on these sites winter oilseed rape plants were adversely affected by fungal pathogens during the experimentation, among which *P. brassicae* was the most important disease. Testing the effect of S fertilisation on the disease incidence and severity of fungal pathogens on these sites, it has been found that S did not induce resistance towards *P. brassicae*, while infections caused by the biotrophic fungus *P. parasitica* were suppressed.
- 6) Changes in metabolite concentrations were found in leaf disc samples that were visually infected by *P. brassicae*. Whereas in Braunschweig infections by *P. brassicae* triggered an increase in the cysteine and GSH content, in Aberdeen a decrease was noted. The results from enzymatic activities suggested that L-cysteine desulphhydrase (LCD), which activity is directly induced by an infection with *P. brassicae*, seems to be the enzyme involved in the release of fungitoxic H₂S.
- 7) A significant positive relationship between the LCD and O-acetylserine(thiol)lyase (OAS-TL) activity was found, indicating that also the activity of OAS-TL is increasing after infection, but not as a direct result of infection rather than as a reaction to the activity of LCD, which is consuming cysteine the product of OAS-TL reaction. Therefore, OAS-TL might participate in increasing the H₂S release in a passive way.
- 8) Regarding the infection of winter oilseed rape by *L. maculans*, standard statistical procedures have failed to reveal interactions between infection and S-containing compounds (GSH, GSLs) under the field trial conditions in Braunschweig. The geostatistical analysis was successfully applied to acquire information about the spatial variability of plant parameters and the probability distribution of *L. maculans* epidemics.
- 9) Glutathione and the risk of severe infections by *L. maculans* displayed spatial autocorrelation, which was quantitatively analysed using variography. The derived semi-variogram models revealed a moderate spatial dependency of 51% and 46% for GSH and the probability of severe *L. maculans* infections, respectively. For the parameters total S and total GSL content no spatial correlations were found.

- 10) The results from the small scale spatial variability revealed that plant parameters and the risk of severe infections by *L. maculans* varied spatially. The two cultivars displayed a different susceptibility against *L. maculans*, with *Bristol* being only slightly affected and *Lipton* being susceptible. Spatial patterns for the probability of severe fungal infections matched that of the S status of the crop in such a way that a higher risk was related to a lower S status and a lower GSL content. Additionally, plants responded to fungal infections with an increased GSH production, whereby the onset of synthesis seemed to be dependent on some threshold infection.

The present research work tried to contribute to the identification of S-containing metabolites governing the pathogenesis in dependence on the S supply under field conditions. Pests and diseases as well as plant physiological parameters have a spatio-temporal distribution. Therefore, in order to identify the mechanisms involved in *SIR*, their interactions and possible triggers, infection-directed sampling strategies and geostatistical analysis were employed. These strategies offered an efficient tool to notice the metabolic changes triggered in plant tissues by the presence of fungal infections and to predict the risk of infections in relation to the S-containing metabolites.

Zusammenfassung: Untersuchungen zur Bedeutung des Einflusses der Schwefel- und Stickstoffversorgung auf schwefelhaltige Stoffwechselprodukte in *Brassica napus* L. für die Schwefel Induzierte Resistenz (*SIR*)

Ziel der vorliegenden Forschungsarbeit war es, den Effekt der S-Ernährung auf S-haltige Metabolite zu untersuchen und diesen in Beziehung zu pilzlichen Infektionen zu setzen. Die Untersuchungen basierten auf der Hypothese, dass über Veränderungen der S-Ernährung der wachsenden Pflanze biochemische Prozesse des primären sowie sekundären S-Metabolismus stimuliert werden, wodurch es zu einer Reduktion und/oder Verzögerung der pilzlichen Infektion kommen kann. Die Bedeutung dieser Arbeit liegt, neben einer Steigerung des Wissens über die Signifikanz von S-haltigen Metaboliten für die pflanzliche Resistenz, in der Entwicklung infektionsabhängiger Beprobungsstrategien und des Einsatzes geostatistischer Verfahren, um ein verbessertes Verständnis zwischen Pilzkrankheiten und individuellen S-Fractionen unter Feldbedingungen zu erreichen.

Die Untersuchungen wurden an drei verschiedenen Standorten durchgeführt, die sich in Klima, Bodentyp und atmosphärischen S-Depositionen unterschieden: Braunschweig (E 10° 27', N 52° 18'), Aberdeen (W 2° 13', N 57° 12') und Inverness (W 4° 5', N 57° 53'). Der S-Bedarf von Winterraps wurde durch Sulfatdüngung über den Boden gedeckt, um einen potentiellen fungiziden Effekt einer Blattdüngung wie bei Zufuhr von Elementarschwefel zu vermeiden. Besondere Aufmerksamkeit wurde den S-haltigen, möglicherweise in die Resistenz involvierten Inhaltsstoffen Cystein, Gluthation (GSH), Glucosinolaten (GSLs) und H₂S freisetzenden Enzymen gewidmet, für die eine Beziehung zum S-Ernährungstatus der Pflanze nachgewiesen wurde.

Die wesentlichen, in dieser Arbeit dargestellten Ergebnisse lassen sich wie folgt zusammenfassen:

- 1) Die S-Düngung steigerte auf allen Standorten den Samenertrag leicht (nicht signifikant), während N-Düngung und Fungizidbehandlung den Samenertrag signifikant erhöhten.
- 2) Die S-Düngung hatte einen positiven signifikanten Effekt auf alle untersuchten S-Fractionen (z.B. Gesamt-S, SO₄-S, Cystein, GSH, GSLs). Dieses ist ein Hinweis dafür, dass S-Düngung zu einer Erhöhung des Potentials der *SIR* führte. Deutliche Unterschiede in den S-Fractionen wurden zwischen den Versuchsstandorten und Jahren festgestellt, was unter anderem ein Indiz für unterschiedliche Verfügbarkeiten des Schwefels ist. Die stärkste

- Wirkung der S-Düngung wurden in Inverness, dem Standort mit der geringsten S-Versorgung, beobachtet.
- 3) Hohe N-Gaben steigerten den Gesamt-N-Gehalt in jungen Blättern und den Gesamt-S- und GSL-Gehalt in Samen.
 - 4) Zwischen beiden Sorten gab es Unterschiede bei den Parametern Cystein- und Progoitringehalt, sowie Samenertrag. Die Sorte *Lipton* wies im Gegensatz zu der Sorte *Bristol* geringere Cystein- und höhere Progoitringehalte sowie höhere Erträge auf.
 - 5) Visuelle Bonituren in Schottland zeigten, dass der Winterraps in diesen Regionen in allen Versuchsjahren negativ durch pilzliche Pathogene beeinflusst wurde, wobei *P. brassicae* der bedeutendste pilzliche Erreger war. Die Prüfung des Einflusses der S-Düngung auf die Befallshäufigkeit und -schwere führte zu dem Ergebnis, dass diese keine Wirkung auf den Pilz *P. brassicae* hatte. Hingegen wurden Infektionen des biotrophen Pilzes *P. parasitica* durch S-Düngung unterdrückt.
 - 6) „Leaf-disc“ Proben, die sichtbar mit *P. brassicae* infiziert waren, wiesen eine Veränderung der Konzentrationen S-haltiger Metabolite auf. Während Infektionen durch *P. brassicae* in Braunschweig zu einer Steigerung der Cystein- und GSH-Gehalte führten, wurde in Aberdeen ein Rückgang beobachtet. Die Ergebnisse der Enzymaktivitäten zeigen, dass das Enzym L-Cysteindesulhydrase (LCD) anscheinend an der Freisetzung von fungitoxischem H₂S beteiligt ist. Die Aktivität dieses Enzyms wurde direkt durch eine Infektion mit *P. brassicae* beeinflusst.
 - 7) Zwischen der Aktivität von LCD und O-Acetylserinthiollyase (OAS-TL) bestand eine signifikant positive Beziehung. Anscheinend wurde die gestiegene Aktivität der OAS-TL nicht direkt durch eine Infektion hervorgerufen. Sie ist eher eine Reaktion auf die veränderte LCD Aktivität. Da LCD Cystein abbaut, könnte OAS-TL an einer steigenden H₂S Freisetzung passiv beteiligt sein.
 - 8) Zwischen Infektionen an Winterraps mit *L. maculans* und S-haltigen, in die Resistenz involvierten Inhaltsstoffen (GSH, GSLs), konnten unter Feldbedingungen in Braunschweig mit Hilfe der standardisierten statistischen Analysen keine Beziehungen nachgewiesen werden. Geostatistische Analysen wurden dagegen erfolgreich eingesetzt, um Informationen

über die räumliche Verteilung pflanzlicher Parameter und die wahrscheinliche Verteilung einer *L. maculans* Epidemie zu erhalten.

- 9) Glutathion und das Risiko eines starken Befalls mit *L. maculans* zeigten räumliche Auto-Korrelationen, die mittels Variogrammanalyse quantitativ erfasst wurden. Die erzeugten Semi-Variogramm-Modelle zeigten eine mittlere räumliche Abhängigkeit mit Werten von 51% und 46% für GSH und das Risiko eines starken Befalls mit *L. maculans*. Für die Parameter Gesamt-S und Gesamt-GSL-Gehalt wurden keine räumlichen Beziehungen gefunden.
- 10) Die Ergebnisse der kleinräumigen Variabilität zeigten, dass Pflanzenmerkmale und das Risiko eines starken Befalls mit *L. maculans* räumlich variierten. Beide Sorten zeigten eine unterschiedliche Empfindlichkeit gegenüber *L. maculans*, wobei *Bristol* nur geringfügig infiziert wurde, *Lipton* hingegen sehr empfindlich reagierte. Räumliche Muster für die Wahrscheinlichkeit starker Infektionen stimmten mit dem S-Status der Pflanzen dahingehend überein, dass das Risiko mit abnehmender S-Versorgung und abnehmendem GSL-Gehalt zunahm. Zudem konnte ein erhöhter GSH-Gehalt als Folge der Infektion festgestellt werden, wobei dieses scheinbar von einem bestimmten Infektionsdruck abhing.

Im Rahmen der vorliegenden Forschungsarbeit wurde versucht ein Beitrag zur Bestimmung der S-haltigen Parameter zu leisten, die die Pathogenese in Abhängigkeit von der S-Düngung unter Feldbedingungen regulieren. Krankheiten und auch pflanzenphysiologische Parameter zeigten hierbei eine räumliche und zeitliche Verteilung. Daher wurden infektionsabhängige Beprobungsstrategien und geostatistische Analysemethoden angewendet, um Mechanismen zu identifizieren, die in *SIR* involviert sind. Darüber hinaus wurden Interaktionen und mögliche Auswirkungen analysiert. Diese Strategien stellten ein effizientes Werkzeug dar, um metabolische Veränderungen, die durch pilzliche Infektionen im pflanzlichen Gewebe ausgelöst wurden, zu erkennen und um darauf aufbauend das Risiko einer Infektion in Abhängigkeit von den S-haltigen fungitoxischen Metaboliten vorherzusagen.

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7 Appendix

Table A.1: Meteorological records during the experimentation years in Braunschweig.

Month	Temperature (°C)			Precipitation (mm)			Sunshine hours (h)		
	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003
August	17.8	19.1	19.6	36	48	101	217	202	207
September	14.5	12.6	13.9	40	149	19	124	69	148
October	11.5	13.1	12.0	33	41	96	109	103	107
November	7.50	5.40	5.40	33	46	105	71	48	45
December	4.10	0.40	33 -0.80		80	81	66	30	46
January	1.50	2.60	0.80	41	45	75	52	67	48
February	2.80	6.00	29 -1.40		74	8	67	88	112
March	3.60	5.80	5.50	64	33	23	73	109	157
April	8.00	8.30	9.00	47	79	26	134	148	214
May	14.4	14.5	14.5	14	55	27	287	185	213
June	14.2	17.0	19.0	96	89	37	165	205	262
July	19.0	17.7	20.5	69	212	49	256	161	282

Table A.2: Meteorological records during the experimentation years in Aberdeen.

Month	Temperature (°C)			Precipitation (mm)			Sunshine hours (h)		
	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003
August	14.714.2	15.0		81.4	88	107.8	n.a.	130	139.3
September	12.4	11.6	12.9	105.8	84	16	n.a.	93.2	126.1
October	9.02	11.9	8.02	120.2	116.8	214.7	n.a.	99.2	102
November	5.54	6.47	7.45	140.2	48	185.4	n.a.	70.7	57.4
December	4.45	3.46	5.41	118.9	73.9	113.6	n.a.	41.6	15.9
January	2.86	4.32	4.24	65	52	94.2	n.a.	56.5	69.2
February	2.58	4.43	3.43	80	65	11.3	n.a.	90.5	76.6
March	3.10	5.61	6.34	105.4	33.8	17.2	n.a.	136.4	191.5
April	7.626.56	8.15		45.8	26.8	55.2	n.a.	165	160.4
May	10.610.9	10.5		21.4	54	86.8	n.a.	197.8	199.1
June	13.111.9	14.2		44.2	107.2	17	n.a.	178.3	195.9
July	13.914.7	16.3		35.4	101	13.2	n.a.	141.9	180.1

note: n.a. – not available

Table A.3: Meteorological records during the experimentation years in Inverness.

Month	Temperature (°C)			Precipitation (mm)			Sunshine hours (h)		
	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003	2000/2001	2001/2002	2002/2003
August	14.614.8	15.3		58	91.5	48.3	n.a.	120	121.1
September	11.513.1	13.0		65.3	57	25.9	n.a.	64.4	104.8
October	11.99.44	8.15		77.4	103.8	118.2	n.a.	77.2	94.6
November	7.225.32	7.02		82.6	66.4	106.3	n.a.	46.5	57.3
December	3.724.28	4.16		61.2	64.4	15.4	n.a.	36.8	32.2
January	5.331.83	3.81		10.8	44.6	53.4	n.a.	45.6	35.6
February	4.483.30	3.60		53.5	117.8	18.7	n.a.	65.9	124.7
March	6.363.62	7.14		51.8	41.1	32.6	n.a.	116.1	176.6
April	8.836.71	9.30		26.7	43.2	31.4	n.a.	160.5	154.2
May	11.111.5	10.9		37.6	30.8	55.2	n.a.	214.3	174.5
June	13.812.1	14.9		48	60.3	32.9	n.a.	160.6	260.4
July	14.213.9	16.2		74.4	96.5	27.7	n.a.	117.3	156

note: n.a. – not available

Table A.4: The completely randomised block design in Braunschweig (e.g. season 2001/2002).

Test factor	Rates	
Cultivar (<i>Bristol; Lipton</i>)	A1	A2
S fertilisation [0; 150 kg ha ⁻¹]	B1	B2
N fertilisation [100; 200 kg ha ⁻¹]	C1	C2
Fungicide (without; with)	D1	D2

A1	A2	A2	A1		A2	A1	A2	A2		A1	A2	A1	A1		A2	A1	A2	A1
B1	B1	B1	B1		B1	B1	B2	B2		B2	B1	B2	B2		B2	B2	B2	B1
C2	C1	C2	C1		C2	C2	C1	C1		C1	C1	C1	C2		C2	C2	C2	C1
D1	D1	D2	D2		D1	D2	D2	D1		D1	D2	D2	D2		D1	D1	D2	D1
16	15	14	13		32	31	30	29		48	47	46	45		64	63	62	61
A2	A1	A2	A1		A1	A2	A1	A1		A2	A2	A1	A2		A1	A2	A1	A2
B2	B2	B1	B1		B2	B2	B2	B2		B1	B2	B1	B1		B1	B1	B1	B2
C2	C1	C1	C1		C2	C2	C1	C2		C1	C1	C2	C2		C2	C2	C1	C1
D1	D1	D2	D1		D1	D2	D2	D2		D1	D1	D1	D1		D2	D2	D2	D2
9	10	11	12		25	26	27	28		41	42	43	44		57	58	59	60
A2	A2	A2	A2		A2	A2	A2	A2		A1	A1	A1	A1		A1	A1	A1	A1
B2	B1	B2	B2		B1	B2	B1	B1		B1	B1	B2	B1		B2	B2	B1	B2
C1	C2	C2	C1		C1	C2	C2	C1		C1	C2	C2	C1		C1	C2	C2	C1
D2	D1	D2	D1		D1	D1	D2	D2		D2	D2	D1	D1		D1	D2	D1	D2
8	7	6	5		24	23	22	21		40	39	38	37		56	55	54	53
A1	A1	A1	A1		A1	A1	A1	A1		A2	A2	A2	A2		A2	A2	A2	A2
B2	B2	B2	B1		B2	B1	B1	B1		B2	B2	B2	B1		B2	B1	B1	B1
C2	C2	C1	C2		C1	C1	C2	C1		C2	C2	C1	C2		C1	C1	C2	C1
D2	D1	D2	D2		D1	D2	D1	D1		D2	D1	D2	D2		D1	D2	D1	D1
1	2	3	4		17	18	19	20		33	34	35	36		49	50	51	52
I					II					III					IV			

Table A.5: The split-plot design in randomised blocks in Scotland (e.g. Aberdeen 2002/2003).

Test factor	Rates	
Cultivar (<i>Bristol; Lipton</i>)	A1	A2
S fertilisation [0; 100 kg ha ⁻¹]	B1	B2
N fertilisation [100; 200 kg ha ⁻¹]	C1	C2
Fungicide (without; with)	D1	D2

D1								I	D2							
A2	A2	A1	A2	A1	A2	A1	A1		A1	A2	A2	A2	A1	A1	A2	A1
B1	B2	B2	B2	B1	B1	B2	B1		B1	B1	B2	B1	B1	B2	B2	B2
C2	C2	C1	C1	C1	C1	C2	C2		C1	C2	C1	C1	C2	C2	C2	C1
1	2	3	4	5	6	7	8		9	10	11	12	13	14	15	16

D1								II	D2							
A2	A2	A1	A2	A1	A2	A1	A1		A2	A2	A1	A1	A2	A1	A2	A1
B1	B1	B2	B2	B1	B2	B2	B1		B2	B2	B2	B2	B1	B1	B1	B1
C2	C1	C2	C1	C2	C2	C1	C1		C2	C1	C1	C2	C2	C1	C1	C2
17	18	19	20	21	22	23	24		25	26	27	28	29	30	31	32

D2								III	D1							
A1	A2	A1	A1	A1	A2	A2	A2		A1	A1	A1	A2	A2	A2	A2	A1
B2	B2	B1	B2	B1	B1	B1	B2		B1	B2	B1	B2	B1	B1	B2	B2
C1	C1	C2	C2	C1	C1	C2	C2		C1	C1	C2	C1	C1	C2	C2	C2
33	34	35	36	37	38	39	40		41	42	43	44	45	46	47	48

D2								IV	D1							
A1	A2	A2	A1	A2	A2	A1	A1		A2	A2	A1	A1	A1	A1	A2	A2
B2	B2	B2	B2	B1	B1	B1	B1		B1	B2	B2	B1	B2	B1	B2	B1
C2	A1	C2	C1	C2	C1	C1	C2		C1	C1	C1	C2	C2	C1	C2	C2
49	50	51	52	53	54	55	56		57	58	59	60	61	62	63	64

Table A.6: Dates of sowing and application of fertilisers and pesticides at the experimental site in Braunschweig.

Treatment	Year					
	2000/2001		2001/2002		2002/2003	
	date	dose/ha	G.S.*	date	dose/ha	G.S.*
Sowing	23.08	6 kg	-	22.08	6 kg	-
Brasan (a.i. clomazone)	25.08	3 L	01	23.08	3 L	01
Harvesan (a.i. flusilazole + carbendazim)	06.11	1 L	14-15	20.11	1 L	14-15
S₁	-	-	-	24.08	40 kg	01
S₂	09.03	75 kg	14-15	01.11	40 kg	12
S₃	04.04	75 kg	30	25.03	70 kg	19
N₁	08.03	50/100 kg	14-15	25.03	50/100 kg	19
N₂	04.04	50/100 hg	30	23.04	50/100 kg	50-53
Ronilan (a.i. vinclozolin)	03.05	1.5 kg	60-63	23.04	1.5 kg	50-53
Verisan (a.i. iprodion)	04.05	3 L	60-63	23.04	3 L	50-53

note: * growing stages according to the BBCH scale (Strauss et al., 1994)

Table A.7: Dates of sowing and application of fertilisers and pesticides at the experimental site in Aberdeen.

Treatment	Year					
	2000/2001		2001/2002		2002/2003	
	date	dose/ha	G.S. ¹	date	dose/ha	G.S. ¹
Sowing	29.08	6 kg	-	28.08	6 kg	-
Butisan (a.i. clomazole)	01.09	1.25 L	01	01.09	1.5 L	01
S₁	-	-	-	11.09	50 kg	02
Autumn Punch C (a.i. flusilazole + carbendazim)	11.04	0.4 L	30	02.11	0.4 L	08
N₁	20.03	100 kg	19	12.03	100 kg	14-15
N₂	04.04	100 kg	30	08.04	100 kg	30
S₂	04.04	100 kg	30	12.03	50 kg	14-15
Spring Punch C (a.i. flusilazole + carbendazim)	27.05	0.5 L ²	60-63	08.04	0.4 L	30
				01.09	6 kg	-
				02.09	2.5 L	01
				11.10	50 kg	08
				28.11	0.4 L	14-15
				03.03	100 kg	14-15
				20.03	100 kg	19
				20.03	50 kg	19
				20.03	0.4 L	19

note: ¹growing stages according to the BBCH scale (Strauss et al., 1994); ²Folicur (a.i. tebuconazol) instead of Punch C

Table A.8: Dates of sowing and application of fertilisers and pesticides at the experimental site in Inverness.

Treatment	Year								
	2000/2001		2001/2002		2002/2003				
	date	dose/ha	G.S.*	date	dose/ha	G.S.*	date	dose/ha	G.S.*
Sowing	29.08	6 kg	-	28.08	6 kg	-	01.09	6 kg	-
Butisan (a.i. clomazone)	02.09	1.5 L	01	01.09	1.5 L	01	02.09	1.25 L	01
S₁	-	-	-	11.09	50 kg	02	08.10	50 kg	08
Autumn Punch C (a.i. flusilazole + carbendazim)	08.01	0.4 L	14-15	02.11	0.4 L	14-15	19.11	0.4 L	14-15
N₁	08.03	100 kg	14-15	12.03	100 kg	14-15	05.03	100 kg	14-15
N₂	05.04	100 kg	30	08.04	100 kg	30	19.03	100 kg	19
S₂	20.03	100 kg	19	12.03	50 kg	14-15	19.03	50 kg	19
Spring Punch C (a.i. flusilazole + carbendazim)	26.04	0.4 L	50-53	08.04	0.4 L	30	19.03	0.4 L	19

note: * growing stages according to the BBCH scale (Strauss et al., 1994)

Table A.9: Mineral nutrient content in leaf and seed samples and the recorded seed yield, Braunschweig, 2000–2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
2000/2001																
0	100	<i>Bristol</i>	with	3.99	53.3	0.72	2.93	12.1	28.4	1.63	17.8	2.96	6.63	4.68	2.83	2.56
0	100	<i>Bristol</i>	with	4.04	55.5	0.62	2.93	10.4	25.8	1.55	13.7	3.01	6.66	4.95	2.69	3.26
0	100	<i>Bristol</i>	with	5.04	56.3	1.62	3.24	11.4	31.6	1.76	16.2	3.09	6.63	5.15	2.76	2.68
0	100	<i>Bristol</i>	with	—	57.9	1.11	3.29	12.0	30.2	1.78	17.4	3.17	6.54	4.11	2.94	2.68
MEAN				—	55.8	1.02	3.10	11.5	29.0	1.68	16.3	3.06	6.62	4.72	2.81	3.07
0	100	<i>Bristol</i>	without	4.84	52.5	0.88	3.49	14.6	28.4	1.98	21.6	3.12	6.98	4.74	2.82	3.45
0	100	<i>Bristol</i>	without	5.08	51.3	1.15	3.65	14.6	29.4	2.09	21.8	2.96	6.66	4.83	2.71	3.66
0	100	<i>Bristol</i>	without	4.25	54.4	1.10	3.24	11.9	28.1	1.74	16.1	2.79	6.79	4.33	2.92	3.02
0	100	<i>Bristol</i>	without	—	48.4	1.46	3.00	16.3	27.7	1.79	24.5	3.15	6.54	4.90	2.72	2.81
MEAN				—	51.6	1.15	3.35	14.33	28.4	1.90	21.0	3.01	6.74	4.70	2.79	3.23
0	100	<i>Lipton</i>	with	4.43	50.1	0.73	3.83	13.5	22.6	1.70	13.8	3.37	6.96	4.74	3.16	3.81
0	100	<i>Lipton</i>	with	4.31	52.6	0.58	4.26	13.8	22.5	1.65	14.6	3.33	6.72	4.71	2.87	3.54
0	100	<i>Lipton</i>	with	3.28	42.8	1.15	2.96	16.8	20.2	1.39	19.5	3.19	6.99	4.63	3.17	2.89
0	100	<i>Lipton</i>	with	—	48.7	1.12	3.57	13.6	21.8	1.52	13.5	3.06	6.70	4.36	2.87	2.89
MEAN				—	48.6	0.90	3.66	14.4	21.8	1.57	15.3	3.24	6.84	4.61	3.02	3.39
0	100	<i>Lipton</i>	without	3.62	51.8	0.72	3.10	12.0	20.9	1.29	11.5	3.21	6.62	4.66	2.94	2.77
0	100	<i>Lipton</i>	without	3.41	47.5	0.47	3.77	13.0	23.0	1.63	17.8	3.00	6.62	4.50	2.94	2.96
0	100	<i>Lipton</i>	without	4.39	50.5	1.12	3.97	13.4	23.5	1.65	13.9	3.14	6.52	4.61	3.05	3.19
0	100	<i>Lipton</i>	without	—	49.9	1.05	3.87	15.3	23.9	1.86	19.3	3.00	6.30	3.94	2.96	3.19
MEAN				—	49.9	0.84	3.68	13.4	22.8	1.61	15.6	3.09	6.51	4.43	2.97	3.08
0	200	<i>Bristol</i>	with	3.56	60.9	0.50	3.18	13.5	30.4	1.63	14.5	2.93	6.60	4.47	2.70	3.14
0	200	<i>Bristol</i>	with	3.44	56.0	0.40	2.81	17.9	24.5	1.49	15.8	3.29	6.44	5.14	2.69	3.96
0	200	<i>Bristol</i>	with	4.33	60.5	1.17	3.74	14.0	31.9	1.89	15.5	3.41	6.55	4.91	2.75	3.33
0	200	<i>Bristol</i>	with	—	61.3	0.62	3.18	12.8	30.0	1.73	14.6	3.14	6.30	4.72	2.74	2.82
MEAN				—	59.7	0.67	3.23	14.6	29.2	1.69	15.1	3.19	6.47	4.81	2.72	3.67
0	200	<i>Bristol</i>	without	3.95	53.7	0.60	3.19	17.6	27.4	1.75	16.9	3.31	6.64	4.73	2.88	2.54
0	200	<i>Bristol</i>	without	3.58	57.8	0.59	2.98	12.7	27.5	1.46	14.3	3.31	6.68	4.88	2.70	3.27
0	200	<i>Bristol</i>	without	3.82	63.9	1.10	3.22	11.6	30.4	1.75	13.3	3.12	6.56	4.28	2.82	2.78
0	200	<i>Bristol</i>	without	—	62.2	0.92	2.80	14.8	25.4	1.74	12.5	3.20	6.17	4.17	2.95	2.78
MEAN				—	59.4	0.80	3.05	14.2	27.7	1.68	14.2	3.24	6.51	4.52	2.84	2.97

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
2000/2001																
0	200	<i>Lipton</i>	with	6.08	49.3	0.32	3.58	18.1	18.6	1.48	5.32	3.41	6.43	4.49	2.84	3.61
0	200	<i>Lipton</i>	with	3.65	55.5	0.17	4.16	13.8	24.6	1.54	12.6	3.44	6.46	4.92	2.90	4.04
0	200	<i>Lipton</i>	with	3.90	54.1	0.93	3.82	16.9	24.5	1.60	13.0	3.47	6.49	4.41	2.85	3.61
0	200	<i>Lipton</i>	with	—	50.8	0.49	3.62	15.4	22.1	1.58	14.5	3.16	6.52	4.43	2.96	3.69
MEAN				—	52.4	0.48	3.80	16.0	22.5	1.55	11.4	3.37	6.48	4.56	2.89	3.74
0	200	<i>Lipton</i>	without	3.72	59.3	0.44	3.74	13.4	23.7	1.48	7.80	3.56	6.47	4.57	2.89	3.27
0	200	<i>Lipton</i>	without	2.72	51.5	0.24	2.91	16.6	20.4	1.27	10.9	3.50	6.29	4.39	2.83	3.57
0	200	<i>Lipton</i>	without	3.53	57.3	0.69	3.35	14.2	24.7	1.42	10.8	3.45	6.25	4.55	2.79	3.79
0	200	<i>Lipton</i>	without	—	64.0	0.66	4.15	11.9	25.2	1.58	8.07	2.99	6.48	4.51	2.86	3.61
MEAN				—	58.0	0.51	3.54	14.0	23.5	1.44	9.39	3.37	6.37	4.51	2.85	3.56
150	100	<i>Bristol</i>	with	13.2	47.6	6.43	3.58	21.2	26.8	2.13	1.03	3.26	7.01	4.76	2.84	3.43
150	100	<i>Bristol</i>	with	9.57	45.6	4.56	3.03	18.2	25.5	1.61	1.47	3.41	6.98	5.15	2.79	3.12
150	100	<i>Bristol</i>	with	10.8	55.0	8.53	2.87	12.7	26.1	1.89	0.16	3.33	6.52	4.84	2.90	3.58
150	100	<i>Bristol</i>	with	11.3	49.2	8.51	3.52	16.5	24.1	1.97	0.83	3.10	6.67	5.22	2.78	3.53
MEAN				—	49.3	7.01	3.25	17.1	25.6	1.90	0.87	3.28	6.79	5.00	2.82	3.49
150	100	<i>Bristol</i>	without	11.6	54.6	5.31	2.91	13.8	26.7	1.90	0.32	3.16	6.81	4.65	2.77	3.22
150	100	<i>Bristol</i>	without	10.5	52.3	5.08	3.09	13.3	25.6	1.80	0.25	3.57	6.73	4.76	2.75	3.82
150	100	<i>Bristol</i>	without	12.4	54.7	5.98	3.35	16.5	25.3	2.40	0.67	3.46	6.71	4.36	2.99	2.67
150	100	<i>Bristol</i>	without	—	55.3	8.39	2.42	11.2	23.1	1.56	0.09	3.30	6.70	5.20	2.85	3.55
MEAN				—	54.2	6.19	2.94	13.7	25.2	1.92	0.33	3.37	6.74	4.74	2.84	3.34
150	100	<i>Lipton</i>	with	8.08	40.2	3.76	3.31	18.3	17.0	1.46	0.98	3.41	6.90	4.68	3.13	3.75
150	100	<i>Lipton</i>	with	9.64	50.5	3.51	3.44	13.1	23.3	1.72	0.66	3.62	6.78	4.58	3.12	3.36
150	100	<i>Lipton</i>	with	9.53	50.3	6.71	3.72	15.9	20.6	1.61	0.94	3.60	6.68	4.66	3.00	2.42
150	100	<i>Lipton</i>	with	—	48.0	5.73	2.76	13.0	19.1	1.26	0.37	3.28	6.72	4.80	3.04	3.64
MEAN				—	47.3	4.93	3.31	15.1	20.0	1.51	0.74	3.48	6.77	4.68	3.07	3.37
150	100	<i>Lipton</i>	without	8.53	51.8	3.74	3.08	13.9	20.3	1.41	0.61	3.43	6.64	4.50	3.07	3.80
150	100	<i>Lipton</i>	without	9.28	48.3	3.08	3.31	13.6	21.2	1.57	0.60	3.56	6.80	4.83	3.02	3.79
150	100	<i>Lipton</i>	without	9.34	43.8	6.32	3.45	20.5	19.0	1.61	1.74	3.48	6.99	4.91	3.16	3.52
150	100	<i>Lipton</i>	without	—	43.7	6.71	3.30	18.2	21.1	1.57	1.54	3.48	6.95	4.77	3.06	3.66
MEAN				—	46.9	4.96	3.29	16.5	20.4	1.54	1.12	3.49	6.84	4.75	3.08	3.69

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	
2000/2001															
150	200	<i>Bristol</i>	with	11.3	59.1	5.41	3.10	14.5	28197	0.3978	6.69	4.49	2.88	2.83	
150	200	<i>Bristol</i>	with	12.5	59.3	4.75	3.96	15.6	29221	0.534	6.88	5.20	2.76	4.33	
150	200	<i>Bristol</i>	with	13.1	60.3	8.73	3.65	15.5	28228	0.2900	5.40	4.32	2.45	3.19	
150	200	<i>Bristol</i>	with	10.6	55.7	8.50	3.45	17.4	28.4	0.9374	6.74	5.28	2.878	—	
MEAN				11.9	58.6	6.84	3.54	15.7	28.7	0.54	6.43	4.82	2.74	3.53	
150	200	<i>Bristol</i>	without	12.1	61.1	5.85	3.62	15.5	312612	0.9473	6.72	4.60	2.86	3.45	
150	200	<i>Bristol</i>	without	10.3	59.7	3.95	3.13	13.8	251675	0.3661	6.66	5.04	2.82	3.91	
150	200	<i>Bristol</i>	without	11.9	57.0	5.20	3.30	15.3	26281	0.3666	6.36	4.72	2.94	3.17	
150	200	<i>Bristol</i>	without	11.8	62.9	8.25	3.59	12.6	31.5	0.3258	6.69	5.00	2.403	—	
MEAN				11.5	60.2	5.81	3.41	14.3	28.9	0.49	6.61	4.84	2.83	3.64	
150	200	<i>Lipton</i>	with	8.11	56.1	3.11	3.68	16.8	201442	0.3980	6.92	5.18	3.07	4.04	
150	200	<i>Lipton</i>	with	10.3	57.6	3.45	3.81	14.6	231861	0.4292	6.60	4.59	3.03	3.81	
150	200	<i>Lipton</i>	with	8.20	51.9	5.77	3.09	19.3	22156	1.9185	6.77	4.52	3.12	3.83	
150	200	<i>Lipton</i>	with	8.37	59.4	5.16	3.77	14.2	24.3	0.4013	6.89	4.80	3.996	—	
MEAN				8.73	56.3	4.37	3.59	16.2	22.8	0.95	6.79	4.77	3.05	3.91	
150	200	<i>Lipton</i>	without	10.0	54.3	3.32	4.15	20.3	211077	0.6782	6.47	4.38	2.96	3.84	
150	200	<i>Lipton</i>	without	8.13	50.7	3.95	3.03	17.1	22187	0.997	6.45	4.22	2.89	4.11	
150	200	<i>Lipton</i>	without	8.38	61.9	3.77	3.27	14.4	211759	0.4916	6.79	4.10	3.26	3.05	
150	200	<i>Lipton</i>	without	9.35	55.4	6.16	3.47	15.6	26.0	0.8990	6.96	5.12	3.392	—	
MEAN				8.97	55.6	4.30	3.48	16.9	22.9	0.75	6.67	4.46	3.05	3.73	
2001/2002															
0	100	<i>Bristol</i>	with	5.44	42.1	1.13	3.99	22.0	21.5	13333	7.24	5.94	2.85	1.08	
0	100	<i>Bristol</i>	with	4.32	37.9	1.01	2.69	21.3	20.0	12621	7.40	5.93	2.99	2.78	
0	100	<i>Bristol</i>	with	4.58	39.1	1.24	3.51	24.1	20.4	14523	7.31	6.10	2.82	1.30	
0	100	<i>Bristol</i>	with	3.81	40.4	0.64	3.30	24.0	19.0	1327	7.61	6.07	3.07	—	
MEAN				4.54	39.9	1.01	3.37	22.8	20.2	13.6	7.39	6.01	2.93	1.86	

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
2001/2002																
0	100	Bristol	without	5.27	45.4	0.96	3.57	25.0	23.9	1.75	15.8	3.29	7.04	5.91	2.75	1.70
0	100	Bristol	with		41.8	0.89	3.43	24.2	22.0	1.55	14.8	3.28	7.20	5.90	2.77	2.35
0	100	Bristol	with		43.6	1.10	3.81	23.3	19.5	1.39	11.4	3.11	7.11	5.97	2.79	1.78
0	100	Bristol	with		43.6	1.00	3.68	24.7	22.4	1.54	13.5	2.82	7.14	6.03	2.75	2.54
	MEAN				43.6	0.99	3.62	24.3	22.0	1.56	13.9	3.13	7.12	5.95	2.76	2.09
0	100	Lipton	26 with		39.9	0.73	3.74	21.3	24.4	1.46	13.6	3.48	6.75	6.08	2.77	2.05
0	100	Lipton	49 with		43.4	0.72	4.25	23.5	22.5	1.58	16.1	3.41	6.96	5.98	2.89	2.47
0	100	Lipton	80 with		40.7	1.03	3.40	20.3	19.5	1.24	12.7	3.32	6.72	5.51	2.71	2.43
0	100	Lipton	85 with		38.9	0.87	3.41	27.7	19.4	1.42	17.7	3.87	6.90	5.86	2.82	1.82
	MEAN				40.7	0.84	3.70	23.2	21.5	1.43	15.0	3.52	6.83	5.86	2.80	2.19
0	100	Lipton	with		41.4	0.93	3.72	24.9	20.7	1.52	14.6	3.36	6.80	5.37	2.75	0.91
0	100	Lipton	with		42.8	0.97	3.10	22.0	20.2	1.23	11.3	3.44	7.09	5.73	2.89	2.41
0	100	Lipton	with		42.6	0.94	3.60	26.6	17.9	1.25	12.5	3.41	6.90	5.56	2.87	1.54
0	100	Lipton	with		44.4	1.09	3.75	23.2	20.7	1.54	12.8	3.49	6.99	5.58	2.85	3.19
	MEAN				42.8	0.98	3.54	24.2	19.9	1.39	12.8	3.43	6.95	5.56	2.84	2.01
0	200	Bristol	46 with		53.5	0.90	3.90	26.5	23.5	1.88	11.4	3.23	6.63	5.43	2.54	1.96
0	200	Bristol	49 with		45.1	0.90	3.53	23.2	21.6	1.57	10.7	3.23	7.03	5.68	2.80	1.23
0	200	Bristol	46 with		48.2	0.95	3.69	22.6	22.1	1.47	10.2	4.19	7.11	5.77	2.93	0.88
0	200	Bristol	41 with		47.6	1.15	3.82	28.0	22.1	1.50	11.3	3.33	6.84	5.85	2.66	1.08
	MEAN				48.6	0.98	3.74	25.0	22.3	1.61	10.9	3.50	6.90	5.68	2.73	1.29
0	200	Bristol	with		54.6	1.11	3.95	25.3	23.6	1.81	8.15	3.40	6.85	5.57	2.71	1.48
0	200	Bristol	with		50.2	0.79	3.13	21.9	23.6	1.51	11.6	3.24	7.07	5.61	2.75	1.39
0	200	Bristol	with		50.1	0.89	4.19	25.8	21.2	1.70	11.2	3.19	6.65	5.12	2.47	0.92
0	200	Bristol	with		49.7	0.65	3.73	23.4	22.1	1.58	9.62	3.23	6.92	5.29	2.75	1.20
	MEAN				51.1	0.86	3.75	24.1	22.6	1.65	10.2	3.27	6.87	5.40	2.67	1.25
0	200	Lipton	27 with		53.0	0.88	4.63	27.1	22.6	1.65	13.2	3.42	6.78	5.53	2.74	1.17
0	200	Lipton	70 with		50.5	0.97	4.06	22.4	23.0	1.68	10.7	3.22	6.79	5.55	2.73	2.30
0	200	Lipton	35 with		49.3	0.90	3.86	23.9	21.5	1.65	9.14	3.38	6.98	5.54	2.85	1.91
0	200	Lipton	36 with		52.8	0.94	4.27	24.6	19.4	1.42	9.21	3.54	7.15	5.75	2.80	2.14
	MEAN				51.4	0.92	4.21	24.5	21.6	1.60	10.6	3.39	6.92	5.59	2.78	1.88

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca
mg g ⁻¹															
2001/2002															
0	200	Lipton	without	5.17	53.6	1.63	4.50	24.4	24.4	1.79	11.8	6.73	5.29	2.62	1.58
0	200	Lipton	with		49.8	0.97	3.89	24.5	22.4	1.60	9.52	6.54	5.59	2.72	0.83
0	200	Lipton	with		49.2	0.90	4.09	26.9	22.1	1.50	10.8	6.85	5.17	2.51	1.63
0	200	Lipton	with		48.4	0.71	3.79	29.0	20.3	1.64	9.83	6.80	5.43	2.82	1.78
MEAN					50.2	1.05	4.07	26.2	22.3	1.63	10.5	6.73	5.37	2.67	1.45
150	100	Bristol	with		41.4	2.30	3.54	20.5	22.7	1.78	1.90	7.00	5.62	2.65	1.81
150	100	Bristol	with		41.6	4.15	3.02	22.3	20.4	1.40	2.16	7.60	6.11	3.08	1.98
150	100	Bristol	with		36.9	1.69	2.97	22.2	19.2	1.36	1.74	7.34	5.47	2.71	1.68
150	100	Bristol	with		37.0	4.11	2.97	22.8	19.6	0.98	1.68	7.39	5.82	2.80	3.23
MEAN					39.2	3.06	3.13	22.0	20.5	1.38	1.87	7.33	5.75	2.81	2.18
150	100	Bristol	with		41.9	2.93	3.54	25.8	21.3	1.68	2.53	7.14	5.90	2.79	1.37
150	100	Bristol	with		39.9	4.58	3.15	22.2	22.6	1.64	2.01	7.46	5.79	2.84	1.13
150	100	Bristol	with		39.4	1.65	2.95	24.4	19.4	1.29	2.22	7.17	5.46	2.85	1.25
150	100	Bristol	with		39.6	1.43	2.92	22.9	20.0	1.31	2.53	7.59	5.93	2.91	0.70
MEAN					40.2	2.65	3.14	23.8	20.8	1.48	2.32	7.34	5.77	2.85	1.11
150	100	Lipton	with		38.7	2.18	3.24	22.3	20.3	1.40	2.81	7.12	5.92	2.95	2.61
150	100	Lipton	with		42.3	4.31	3.14	20.5	18.3	1.25	1.81	6.98	6.00	3.04	3.20
150	100	Lipton	with		36.2	1.76	3.33	21.7	16.9	1.55	2.05	7.28	5.74	3.09	2.94
150	100	Lipton	with		38.9	5.76	3.55	22.8	20.2	1.43	3.17	8.20	6.60	3.39	3.95
MEAN					39.0	3.50	3.32	21.8	18.9	1.41	2.46	7.39	6.07	3.12	3.18
150	100	Lipton	with		39.7	1.26	3.26	23.0	21.7	1.56	3.10	7.07	5.22	2.77	2.13
150	100	Lipton	with		40.1	5.23	3.34	23.3	17.6	1.27	2.97	6.87	5.33	2.94	1.17
150	100	Lipton	with		37.9	3.07	3.30	22.8	20.4	1.46	2.52	7.12	5.28	2.88	2.17
150	100	Lipton	with		39.8	1.12	3.38	23.7	21.5	1.40	3.29	6.79	5.62	2.79	1.63
MEAN					39.4	2.67	3.32	23.2	20.3	1.42	2.97	6.96	5.36	2.85	1.78
150	200	Bristol	with		51.6	1.50	3.85	24.5	22.8	1.81	1.79	7.06	5.34	2.68	1.55
150	200	Bristol	with		51.4	1.33	4.16	24.8	21.4	1.85	1.41	7.12	5.85	2.93	2.47
150	200	Bristol	with		49.9	3.20	3.19	24.1	19.2	1.47	1.46	7.00	5.36	2.52	1.59
150	200	Bristol	with		45.0	1.54	3.43	24.1	19.3	1.44	1.28	6.99	5.69	2.72	1.75
MEAN					49.5	1.89	3.66	24.4	20.7	1.64	1.48	7.04	5.56	2.71	1.84

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	
2001/2002															
150	200	<i>Bristol</i>	without	10.4	53.9	2.16	4.02	26.6	24.9	2.16	2.42	7.04	5.28	2.74	0.76
150	200	<i>Bristol</i>	without	7.70	51.3	1.08	2.93	23.8	21.4	1.40	1.40	7.09	5.83	2.86	1.25
150	200	<i>Bristol</i>	without	9.01	50.8	1.89	3.44	25.0	20.5	1.78	1.53	7.44	6.29	2.99	0.88
150	200	<i>Bristol</i>	without	8.66	54.4	2.22	3.45	25.9	20.8	0.60	1.40	6.94	5.62	2.80	0.98
MEAN				8.95	52.6	1.84	3.46	25.3	21.9	1.49	1.84	7.13	5.75	2.85	0.97
150	200	<i>Lipton</i>	with	8.27	50.0	1.61	4.07	26.0	23.8	1.77	2.39	6.74	5.42	2.72	2.55
150	200	<i>Lipton</i>	with	9.51	44.8	1.60	3.19	22.9	20.2	1.38	1.49	6.90	5.65	2.92	1.46
150	200	<i>Lipton</i>	with	9.96	47.9	2.34	3.84	24.9	21.7	1.72	2.39	6.78	5.64	2.91	2.08
150	200	<i>Lipton</i>	with	9.68	48.3	1.91	3.71	24.9	21.2	1.62	2.14	6.67	5.63	2.86	2.93
150	200	<i>Lipton</i>	without	8.87	48.5	2.43	3.51	25.8	21.6	1.72	3.49	6.83	5.32	2.90	2.52
150	200	<i>Lipton</i>	without	10.4	48.9	1.89	4.03	25.3	22.5	1.91	2.69	6.96	5.55	3.01	1.49
150	200	<i>Lipton</i>	without	9.27	53.1	1.52	3.80	26.3	22.8	1.51	1.43	6.94	5.52	3.09	1.45
MEAN				9.51	50.5	1.95	3.87	26.7	22.0	1.71	2.57	6.75	5.57	2.82	1.50
2002/2003															
0	100	<i>Bristol</i>	with	2.91	37.6	0.44	3.06	29.7	15.5	1.50	12.68	6.40	3.67	2.78	3.07
0	100	<i>Bristol</i>	with	3.17	34.9	n.a.	3.04	21.6	15.4	1.34	12.47	6.67	4.03	2.91	3.35
0	100	<i>Bristol</i>	with	3.13	33.1	0.42	3.28	24.5	14.8	1.53	12.37	6.21	4.04	2.87	3.16
0	100	<i>Bristol</i>	with	3.35	34.7	0.35	3.05	29.9	16.3	1.45	10.99	6.31	3.87	2.81	2.65
MEAN				3.14	35.1	0.40	3.11	26.4	15.5	1.46	12.3	6.40	3.90	2.84	3.06
0	100	<i>Bristol</i>	without	2.87	36.4	0.30	2.88	28.0	15.3	1.40	12.67	6.21	3.63	2.81	3.08
0	100	<i>Bristol</i>	without	3.39	36.9	0.79	3.45	20.4	18.0	1.58	18.67	6.24	3.89	2.79	2.75
0	100	<i>Bristol</i>	without	2.95	35.7	n.a.	2.91	29.1	14.1	1.48	12.74	6.44	3.52	2.82	2.56
0	100	<i>Bristol</i>	without	2.88	36.4	0.20	2.96	28.6	15.5	1.61	12.55	5.93	3.52	2.73	2.76
MEAN				3.02	36.4	0.43	3.05	26.5	15.7	1.52	14.9	6.20	3.64	2.79	2.79

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
mg g ⁻¹																
2002/2003																
0	100	Lipton	with	2.79	36.2	0.45	2.97	29.0	13.5	1.31	13.9	3.00	6.22	3.61	2.81	3.23
0	100	Lipton	42 with		32.5	0.24	2.97	18.5	10.9	1.02	8.78	3.00	6.44	3.93	2.86	2.75
0	100	Lipton	74 with		34.3	0.20	3.17	25.0	12.8	1.23	9.39	2.98	6.76	4.06	2.98	3.09
0	100	Lipton	55 with		33.8	n.a.	3.03	23.3	12.2	1.23	15.5	3.07	6.71	3.90	3.00	2.91
	MEAN				34.2	0.29	3.04	24.0	12.4	1.20	11.9	3.01	6.53	3.88	2.91	3.00
0	100	Lipton	with		39.6	0.27	3.66	25.6	14.3	1.50	9.51	2.96	6.24	3.56	2.84	2.86
0	100	Lipton	with		35.7	n.a.	2.93	26.3	12.5	1.09	11.9	3.01	6.33	3.76	2.79	3.20
0	100	Lipton	with		33.1	0.29	2.97	28.0	13.7	1.31	11.7	3.08	6.32	3.84	2.88	3.18
0	100	Lipton	with		37.1	0.22	3.10	27.1	12.1	1.29	13.6	3.12	6.28	3.76	2.89	3.03
	MEAN				36.4	0.26	3.17	26.7	13.1	1.30	11.7	3.04	6.29	3.73	2.85	3.07
0	200	Bristol	23 with		44.2	0.06	3.92	28.7	18.0	2.05	19.3	2.62	6.23	3.71	2.78	3.03
0	200	Bristol	88 with		39.9	0.18	2.83	31.5	15.8	1.67	11.4	2.82	6.21	3.77	2.84	3.04
0	200	Bristol	37 with		37.4	n.a.	2.68	29.4	15.2	1.28	12.1	2.67	6.26	4.10	2.80	2.96
0	200	Bristol	66 with		35.7	0.30	2.75	29.7	14.2	1.51	10.7	2.71	6.60	3.58	2.80	2.72
	MEAN				39.3	0.18	3.05	29.8	15.8	1.63	13.4	2.70	6.32	3.79	2.80	2.94
0	200	Bristol	with		47.1	0.65	3.79	19.9	20.1	1.87	10.1	2.52	6.22	3.63	2.75	2.60
0	200	Bristol	with		41.3	0.56	3.04	20.1	16.7	1.44	10.6	2.68	6.09	3.76	2.80	2.72
0	200	Bristol	with		35.9	0.24	2.91	29.2	15.1	1.47	7.95	2.66	6.22	3.68	2.83	2.92
0	200	Bristol	with		37.5	0.11	3.12	29.0	15.2	1.52	10.9	2.79	6.38	3.47	2.71	3.03
	MEAN				40.5	0.39	3.22	24.5	16.8	1.58	9.89	2.66	6.23	3.63	2.77	2.82
0	200	Lipton	53 with		38.3	n.a.	2.71	29.6	13.7	1.39	11.0	3.09	6.37	3.51	2.73	3.18
0	200	Lipton	14 with		40.8	n.a.	3.15	28.5	12.1	1.27	11.4	3.18	6.39	3.91	2.92	2.47
0	200	Lipton	60 with		38.5	0.16	3.10	28.1	11.7	1.27	12.9	2.96	6.62	3.66	2.89	2.63
0	200	Lipton	70 with		36.6	0.30	2.74	30.8	13.7	1.44	10.2	3.10	6.31	3.75	2.82	3.25
	MEAN				38.5	0.23	2.93	29.2	12.8	1.34	11.4	3.08	6.42	3.71	2.84	2.88
0	200	Lipton	with		43.4	0.09	3.53	29.1	15.6	1.89	11.7	3.07	6.28	3.32	2.93	3.29
0	200	Lipton	with		40.8	n.a.	3.29	32.0	15.1	1.53	11.4	3.14	6.27	3.77	2.83	2.90
0	200	Lipton	with		36.3	0.82	2.91	27.5	12.2	1.26	7.50	3.02	6.48	3.67	2.86	2.89
0	200	Lipton	with		39.2	0.49	2.95	22.5	11.8	1.17	6.76	3.04	6.50	3.71	2.82	3.20
	MEAN				39.9	0.47	3.17	27.7	13.7	1.46	9.33	3.07	6.38	3.62	2.86	3.07

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
2002/2003																
150	100	<i>Bristol</i>	with	6.61	36.8	n.a.	2.76	22.0	14.5	1.34	0.61	3.56	6.20	3.60	2.90	3.12
150	100	<i>Bris6094</i>	with		39.6	0.71	2.94	31.5	15.0	1.39	1.52	3.82	6.24	3.45	2.81	2.60
150	100	<i>Bris4058</i>	with		35.2	0.36	2.71	27.1	13.5	1.27	1.57	3.55	6.66	3.91	2.91	3.15
150	100	<i>Bris6097</i>	with		33.6	n.a.	2.66	27.1	12.7	1.33	1.3088		6.15	3.72	2.79	3.07
MEAN				6.03	36.3	0.54	2.77	26.9	13.9	1.33	1.20	3.60	6.31	3.67	2.85	2.98
150	100	<i>Bristol</i>	without		39.5	0.65	3.25	27.1	16.2	1.82	1.59	3.50	6.05	3.36	2.84	2.43
150	100	<i>Bristol</i>	without		32.7	2.50	2.86	17.7	15.8	1.25	0.51	3.34	6.43	3.94	2.80	3.03
150	100	<i>Bristol</i>	without		37.0	0.19	3.11	28.3	16.2	1.51	8.62	3.02	6.24	3.72	2.72	2.81
150	100	<i>Bristol</i>	without		36.9	0.63	3.08	33.1	16.0	1.56	2.3108		6.57	3.38	2.90	2.88
MEAN				6.01	36.5	0.99	3.08	26.5	16.0	1.54	3.20	3.36	6.32	3.60	2.81	2.79
150	100	<i>Lipt6161</i>	with		39.5	0.21	3.56	17.1	13.5	1.34	0.69	3.66	6.75	3.78	3.01	3.05
150	100	<i>Lipt6101</i>	with		41.5	0.57	3.14	33.7	16.0	1.61	3.97	4.04	6.31	3.50	2.97	2.52
150	100	<i>Lipt6164</i>	with		35.3	0.49	3.39	25.1	12.2	1.24	0.92	3.56	6.53	3.76	2.89	3.10
150	100	<i>Lipt6184</i>	with		37.1	0.72	3.1	30.4	13.3	1.48	2.3645		6.40	3.67	2.93	2.59
MEAN				5.98	38.3	0.50	3.30	26.6	13.7	1.42	2.06	3.73	6.50	3.68	2.95	2.82
150	100	<i>Lipton</i>	without		32.5	0.80	2.98	19.0	11.0	1.06	0.78	3.60	6.33	3.54	2.91	2.76
150	100	<i>Lipton</i>	without		39.4	0.36	2.97	24.4	12.1	1.22	1.70	3.77	6.30	3.76	2.92	2.84
150	100	<i>Lipton</i>	without		31.9	0.53	2.68	25.1	12.9	1.07	1.38	3.88	6.22	3.72	2.88	2.98
150	100	<i>Lipton</i>	without		30.7	0.68	2.78	20.2	10.3	1.06	1.3202		6.48	3.53	2.94	2.80
MEAN				4.67	33.6	0.59	2.85	22.2	11.6	1.10	1.29	3.72	6.33	3.64	2.91	2.85
150	200	<i>Bris4044</i>	with		47.8	n.a.	3.60	20.7	19.7	1.84	12.4	2.99	6.38	3.89	2.85	3.02
150	200	<i>Bris4065</i>	with		36.1	0.58	2.56	26.5	13.4	1.13	0.86	3.67	5.95	3.87	2.78	3.04
150	200	<i>Bris6057</i>	with		39.3	0.85	3.11	33.0	16.1	1.76	1.89	3.75	5.87	3.40	2.87	3.37
150	200	<i>Bris6068</i>	with		40.9	0.61	2.92	31.4	14.9	1.68	1.3265		6.26	3.59	2.92	3.14
MEAN				5.34	41.0	0.68	3.05	27.9	16.0	1.60	4.10	3.52	6.12	3.69	2.86	3.14
150	200	<i>Bristol</i>	without		45.4	0.25	2.77	32.9	16.0	1.68	1.95	3.67	5.93	3.43	2.85	2.76
150	200	<i>Bristol</i>	without		43.4	0.73	2.64	31.7	16.8	1.34	1.90	3.51	5.79	3.46	2.71	3.06
150	200	<i>Bristol</i>	without		38.4	1.27	2.75	26.1	14.4	1.28	0.88	3.49	5.67	3.45	2.66	3.01
150	200	<i>Bristol</i>	without		37.6	1.24	2.89	31.9	18.3	1.64	2.3378		5.96	3.55	2.77	2.91
MEAN				5.84	41.2	0.87	2.76	30.6	16.4	1.49	1.77	3.61	5.84	3.48	2.75	2.94

Table A.9: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
mg g ⁻¹																
2002/2003																
150	200	<i>Lipton</i>	with	5.23	42.6	0.18	2.84	26.2	11.5	1.16	0.72	3.84	5.99	3.60	2.86	2.97
150	200	<i>Lipton</i>	with	4.81	40.7	1.24	2.98	28.9	12.5	1.33	1.39	3.81	5.99	3.69	2.94	3.39
150	200	<i>Lipton</i>	with	5.75	45.9	0.81	3.38	19.8	11.6	1.38	0.31	3.72	6.18	3.54	2.95	3.12
150	200	<i>Lipton</i>	with	—	42.2	0.62	2.54	28.6	14.4	1.25	2.56	4.01	5.90	3.72	2.84	2.76
MEAN				—	42.8	0.71	2.94	25.9	12.5	1.28	1.24	3.84	6.02	3.64	2.90	3.06
150	200	<i>Lipton</i>	without	5.75	47.4	n.a.	3.90	21.2	14.6	1.51	0.60	3.73	5.78	3.44	2.75	2.84
150	200	<i>Lipton</i>	without	5.71	43.4	n.a.	3.46	25.5	12.5	1.46	1.01	3.83	5.70	3.49	2.90	3.08
150	200	<i>Lipton</i>	without	6.15	43.5	n.a.	3.38	19.9	14.3	1.32	0.70	3.82	6.28	3.76	2.86	2.92
150	200	<i>Lipton</i>	without	—	38.6	0.50	3.13	26.3	11.4	1.22	1.36	4.01	5.91	3.48	2.96	2.93
MEAN				5.47	43.2	0.50	3.47	23.2	13.2	1.38	0.91	3.84	5.92	3.54	2.87	2.94

note: n.a. – not available

Table A.10: Mineral nutrient content in leaf and seed samples and the recorded seed yield, Aberdeen, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
2000/2001																
0	100	<i>Bristol</i>	with	5.61	49.1	2.28	2.80	28.9	29.8	1.24	23.3	2.60	5.35	4.35	2.44	3.02
0	100	<i>Bristol</i>	with	6.24	53.5	n.a.	3.33	32.9	28.9	1.36	19.3	2.69	5.23	4.82	2.51	2.81
0	100	<i>Bristol</i>	with	6.27	52.1	2.37	3.37	32.5	30.8	1.40	21.5	2.62	5.06	4.63	2.35	3.28
0	100	<i>Bristol</i>	with	—	51.7	n.a.	3.11	29.6	29.3	1.27	19.1	2.65	5.26	4.36	2.45	3.04
MEAN				—	51.6	2.32	3.15	31.0	29.7	1.32	20.8	2.64	5.23	4.54	2.44	3.04
0	100	<i>Bristol</i>	without	5.74	50.1	4.82	3.05	28.3	28.2	1.25	22.0	2.72	5.30	4.47	2.45	2.83
0	100	<i>Bristol</i>	without	4.35	49.2	4.26	2.29	27.5	26.8	1.05	18.7	2.70	5.01	4.78	2.40	3.09
0	100	<i>Bristol</i>	without	5.02	52.6	2.42	2.85	26.1	28.0	1.18	16.2	2.56	5.13	4.48	2.35	3.07
0	100	<i>Bristol</i>	without	—	52.4	2.81	2.88	25.1	28.8	1.19	17.8	2.77	5.17	4.61	2.40	3.16
MEAN				—	51.1	3.58	2.77	26.7	28.0	1.17	18.7	2.69	5.15	4.59	2.40	3.04
0	100	<i>Lipton</i>	with	5.29	50.9	2.64	3.20	25.1	22.9	1.14	20.2	2.57	5.52	4.62	2.68	3.45
0	100	<i>Lipton</i>	with	7.48	52.5	2.61	4.22	30.9	25.9	1.38	20.6	2.63	5.35	4.75	2.64	3.95
0	100	<i>Lipton</i>	with	6.54	50.5	3.74	3.35	29.9	24.0	1.13	18.7	2.62	5.45	4.55	2.55	3.56
0	100	<i>Lipton</i>	with	—	51.5	n.a.	3.82	27.3	24.1	1.36	17.5	2.49	5.23	4.71	2.47	3.51
MEAN				—	51.4	2.99	3.65	28.3	24.2	1.25	19.2	2.58	5.39	4.66	2.59	3.62
0	100	<i>Lipton</i>	without	5.81	48.0	n.a.	3.11	24.6	23.1	1.12	19.0	2.71	5.57	4.71	2.65	3.01
0	100	<i>Lipton</i>	without	7.47	53.0	6.00	4.04	28.7	26.6	1.33	18.8	2.77	5.35	4.35	2.59	3.23
0	100	<i>Lipton</i>	without	6.58	52.6	n.a.	3.54	28.5	25.4	1.31	15.9	2.69	5.18	4.63	2.57	2.87
0	100	<i>Lipton</i>	without	—	51.0	3.80	3.97	26.1	25.1	1.23	19.8	2.75	5.31	4.50	2.51	3.46
MEAN				—	51.1	4.90	3.67	27.0	25.1	1.25	18.4	2.73	5.35	4.54	2.58	3.14
0	200	<i>Bristol</i>	with	4.66	51.0	1.53	2.64	29.8	28.9	1.28	18.8	2.72	4.58	4.23	2.43	3.79
0	200	<i>Bristol</i>	with	5.71	55.1	4.25	3.09	31.2	28.0	1.39	14.2	2.78	5.02	4.68	2.55	3.51
0	200	<i>Bristol</i>	with	6.29	54.2	3.37	3.07	28.9	29.8	1.28	14.6	2.79	4.89	4.66	2.45	3.76
0	200	<i>Bristol</i>	with	—	55.7	3.82	3.22	31.6	28.8	1.40	15.5	2.69	5.16	4.48	2.53	3.29
MEAN				—	54.0	3.24	3.01	30.4	28.9	1.34	15.8	2.75	4.91	4.51	2.49	3.59
0	200	<i>Bristol</i>	without	6.69	53.3	3.74	3.40	33.4	33.6	1.53	17.8	2.81	4.96	4.37	2.46	3.74
0	200	<i>Bristol</i>	without	10.4	55.0	4.09	5.09	42.8	42.2	2.11	21.5	2.83	4.71	4.89	2.40	3.44
0	200	<i>Bristol</i>	without	9.49	47.0	2.69	2.83	29.6	26.3	1.00	5.66	2.67	4.51	4.54	2.37	3.75
0	200	<i>Bristol</i>	without	—	55.1	2.62	2.85	24.4	27.4	1.15	10.5	2.69	4.52	4.73	2.25	3.59
MEAN				—	52.6	3.28	3.54	32.6	32.4	1.45	13.9	2.75	4.68	4.63	2.37	3.63

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds						Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg	
2000/2001																
0	200	<i>Lipton</i>	with	4.63	55.0	1.99	3.17	26.4	23.2	1.06	12.2	2.85	4.97	4.48	2.56	4.30
0	200	<i>Lipton</i>	with	5.72	56.7	3.72	3.05	32.1	28.6	1.39	14.6	2.83	5.12	4.46	2.60	4.35
0	200	<i>Lipton</i>	with	5.61	55.6	2.70	3.37	28.2	26.3	1.23	16.0	2.84	4.95	4.69	2.51	4.46
0	200	<i>Lipton</i>	with	—	<u>55.3</u>	n.a.	<u>3.45</u>	<u>26.6</u>	<u>25.1</u>	<u>1.16</u>	<u>17.2</u>	—	<u>5.22</u>	<u>3.97</u>	<u>2.46</u>	<u>4.41</u>
MEAN				5.50	55.6	2.80	3.26	28.3	25.8	1.21	13.5	2.81	5.07	4.40	2.53	4.38
0	200	<i>Lipton</i>	without	6.52	55.4	n.a.	3.79	28.1	25.8	1.33	12.1	2.96	5.09	4.49	2.58	4.26
0	200	<i>Lipton</i>	without	6.22	54.2	4.14	3.26	27.9	25.7	1.29	11.8	2.97	4.94	4.43	2.53	4.21
0	200	<i>Lipton</i>	without	6.11	54.7	3.58	3.69	30.8	26.3	1.36	16.2	2.88	4.76	4.90	2.51	4.03
0	200	<i>Lipton</i>	without	—	<u>55.1</u>	<u>3.79</u>	<u>3.44</u>	<u>26.7</u>	<u>26.6</u>	<u>1.18</u>	<u>13.6</u>	—	<u>4.88</u>	<u>4.47</u>	<u>2.51</u>	<u>4.32</u>
MEAN				6.24	54.8	3.84	3.55	28.4	26.1	1.29	13.2	3.11	4.92	4.57	2.53	4.21
100	100	<i>Bristol</i>	with	9.62	47.3	8.05	2.87	29.8	25.2	1.10	3.64	2.71	5.44	4.25	2.42	2.70
100	100	<i>Bristol</i>	with	9.90	54.4	6.94	3.15	33.1	26.0	1.25	3.66	2.84	5.59	4.57	2.63	3.39
100	100	<i>Bristol</i>	with	10.6	50.6	7.28	3.27	31.0	29.4	1.26	3.81	2.98	5.54	4.57	2.56	3.27
100	100	<i>Bristol</i>	with	<u>10.3</u>	<u>52.1</u>	n.a.	<u>3.27</u>	<u>34.3</u>	<u>27.4</u>	<u>1.20</u>	<u>3.79</u>	—	<u>5.34</u>	<u>4.18</u>	<u>2.43</u>	<u>3.34</u>
MEAN				10.1	51.1	7.43	3.14	32.1	27.0	1.20	3.71	2.83	5.48	4.39	2.51	3.18
100	100	<i>Bristol</i>	without	11.7	53.8	8.10	3.68	30.2	29.5	1.39	3.44	2.97	5.51	4.52	2.56	2.76
100	100	<i>Bristol</i>	without	8.66	50.8	n.a.	2.77	31.7	27.8	1.15	4.69	2.84	5.20	4.48	2.51	3.09
100	100	<i>Bristol</i>	without	5.75	56.0	7.66	3.37	27.2	31.8	1.40	16.2	2.81	5.76	4.59	2.51	2.18
100	100	<i>Bristol</i>	without	<u>10.5</u>	<u>50.0</u>	<u>9.22</u>	<u>3.14</u>	<u>30.6</u>	<u>28.4</u>	<u>1.25</u>	<u>3.40</u>	—	<u>5.28</u>	<u>4.45</u>	<u>2.45</u>	<u>2.80</u>
MEAN				9.15	52.6	8.33	3.24	29.9	29.4	1.30	7.19	2.95	5.44	4.51	2.51	2.71
100	100	<i>Lipton</i>	with	9.79	44.4	5.60	3.14	30.5	20.1	0.99	3.24	2.64	5.65	4.33	2.52	3.02
100	100	<i>Lipton</i>	with	8.67	51.7	6.18	3.52	28.6	21.0	1.05	2.85	2.62	5.63	4.77	2.74	3.85
100	100	<i>Lipton</i>	with	10.7	49.4	5.13	3.83	29.9	23.0	1.12	2.72	2.60	5.76	4.62	2.68	3.36
100	100	<i>Lipton</i>	with	—	<u>50.2</u>	n.a.	<u>3.06</u>	<u>28.0</u>	<u>19.6</u>	<u>0.99</u>	<u>2.66</u>	—	<u>5.70</u>	<u>4.48</u>	<u>2.53</u>	<u>3.56</u>
MEAN				9.41	48.9	5.63	3.39	29.2	20.9	1.04	2.87	2.59	5.68	4.55	2.62	3.45
100	100	<i>Lipton</i>	without	9.01	49.6	6.82	3.38	22.3	22.5	1.08	2.08	2.80	5.63	4.56	2.59	3.03
100	100	<i>Lipton</i>	without	8.83	50.3	n.a.	3.28	30.5	21.5	1.06	3.72	2.89	5.55	4.75	2.68	3.34
100	100	<i>Lipton</i>	without	9.84	53.3	n.a.	3.60	30.3	23.9	1.17	2.62	2.75	5.51	4.67	2.55	2.99
100	100	<i>Lipton</i>	without	<u>10.8</u>	<u>51.2</u>	<u>6.66</u>	<u>3.84</u>	<u>31.3</u>	<u>25.1</u>	<u>1.24</u>	<u>3.43</u>	—	<u>5.44</u>	<u>4.72</u>	<u>2.49</u>	<u>3.28</u>
MEAN				9.61	51.1	6.74	3.53	28.6	23.2	1.14	2.96	2.79	5.53	4.68	2.58	3.16

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds						Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg	
														mg g ⁻¹		
2000/2001																
100	200	<i>Bristol</i>	with	7.88	53.0	2.02	2.78	30.5	28.7	1.20	4.20	3.14	4.94	4.25	2.45	3.67
100	200	<i>Bristol</i>	with	8.98	57.0	n.a.	3.22	35.3	28.5	1.43	3.21	3.11	5.28	4.60	2.57	3.79
100	200	<i>Bristol</i>	with	8.92	56.4	5.56	3.10	30.3	29.7	1.34	2.91	3.09	5.01	4.38	2.54	3.99
100	200	<i>Bristol</i>	with	—	57.6	7.36	3.32	34.7	30.4	1.38	3.41	2.96	4.87	4.44	2.42	4.14
MEAN				—	56.0	4.98	3.11	32.7	29.3	1.34	3.43	3.07	5.02	4.42	2.50	3.90
100	200	<i>Bristol</i>	without	8.05	53.5	7.16	2.57	31.8	29.9	1.21	4.09	3.18	5.02	4.58	2.54	3.85
100	200	<i>Bristol</i>	without	7.67	55.2	7.18	2.71	32.3	27.5	1.18	3.93	3.31	4.78	4.40	2.50	3.08
100	200	<i>Bristol</i>	without	8.84	53.7	6.07	2.82	31.9	31.3	1.29	4.56	3.09	4.65	4.52	2.49	3.06
100	200	<i>Bristol</i>	without	10.8	55.3	5.94	3.57	29.5	32.0	1.40	2.78	3.83	4.63	4.47	2.42	3.75
MEAN				—	54.4	6.59	2.92	31.4	30.2	1.27	3.84	3.35	4.77	4.49	2.49	3.44
100	200	<i>Lipton</i>	with	7.89	54.6	n.a.	3.25	32.4	26.2	1.26	3.32	2.94	5.11	4.27	2.58	4.50
100	200	<i>Lipton</i>	with	9.18	57.3	n.a.	4.10	40.1	26.9	1.42	4.65	3.03	5.20	4.56	2.67	4.63
100	200	<i>Lipton</i>	with	7.21	55.3	5.99	3.20	29.7	23.7	1.16	2.91	2.97	5.13	4.49	2.57	4.83
100	200	<i>Lipton</i>	with	—	56.4	3.92	3.47	31.1	23.3	1.21	3.15	2.97	5.19	4.37	2.56	4.57
MEAN				—	55.9	4.96	3.51	33.3	25.0	1.26	3.51	2.98	5.16	4.42	2.59	4.63
100	200	<i>Lipton</i>	without	8.11	56.0	5.51	3.39	34.1	24.7	1.33	3.74	3.10	5.03	4.48	2.51	4.47
100	200	<i>Lipton</i>	without	7.95	55.6	n.a.	3.39	31.9	25.6	1.33	3.84	3.08	4.96	4.34	2.56	4.52
100	200	<i>Lipton</i>	without	8.15	56.7	4.14	3.36	32.7	26.0	1.22	3.21	3.11	4.96	4.63	2.46	4.43
100	200	<i>Lipton</i>	without	10.6	56.9	n.a.	4.65	40.8	31.1	1.53	4.19	3.83	5.02	4.39	2.52	4.12
MEAN				—	56.3	4.83	3.70	34.9	26.8	1.35	3.74	3.28	4.99	4.46	2.51	4.39
2001/2002																
0	100	<i>Bristol</i>	with	4.45	41.2	0.79	3.09	26.2	16.7	1.28	27.1	2.72	6.12	5.28	2.47	3.08
0	100	<i>Bristol</i>	with	3.90	44.3	0.60	3.00	25.7	16.8	1.25	26.7	2.95	5.93	4.71	2.42	2.31
0	100	<i>Bristol</i>	with	3.88	50.2	0.52	3.21	24.0	17.6	1.15	20.9	2.89	5.65	5.34	2.54	2.65
0	100	<i>Bristol</i>	with	—	45.0	0.64	2.76	26.0	15.9	1.09	18.6	2.90	5.82	4.93	2.49	2.79
MEAN				4.24	45.2	0.64	3.02	25.5	16.8	1.19	23.3	2.87	5.88	5.06	2.48	2.71

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg			
2001/2002																		
0	100	<i>Bristol</i>	without	4.19	49.7	0.59	2.74	25.6	14.8	1.02	18.9	3.11	6.18	5.03	2.46	2.31		
0	100	<i>Bristol</i>	without	4.10	49.7	1.00	3.63	29.5	20.0	1.24	2.31	3.69	5.87	5.16	2.47	3.36		
0	100	<i>Bristol</i>	without	5.26	42.8	0.67	3.06	25.2	17.9	1.14	15.4	3.43	6.01	5.27	2.36	2.22		
0	100	<i>Bristol</i>	with	—	47.5	0.86	2.99	28.9	16.4	1.08	16.4	2.99	5.90	5.09	2.50	2.53		
	MEAN			4.55	47.4	0.78	3.11	27.3	17.2	1.12	13.2	3.30	5.99	5.14	2.45	2.61		
0	100	<i>Lipton</i>	with	4.07	41.0	0.71	3.20	26.8	14.5	1.13	25.1	2.80	5.85	4.89	2.53	2.85		
0	100	<i>Lipton</i>	with	3.76	50.2	0.43	3.19	26.0	13.6	1.04	21.1	2.93	5.59	4.54	2.47	2.83		
0	100	<i>Lipton</i>	with	3.87	46.2	0.43	3.00	27.2	14.7	0.96	19.4	2.99	5.67	5.02	2.46	1.97		
0	100	<i>Lipton</i>	with	—	44.9	0.65	3.55	23.8	13.8	1.19	11.6	2.85	5.72	4.92	2.43	3.03		
	MEAN			4.26	45.6	0.56	3.24	25.9	14.1	1.08	19.3	2.89	5.71	4.84	2.47	2.67		
0	100	<i>Lipton</i>	without	4.49	40.9	0.94	3.41	27.4	14.7	1.12	18.5	3.00	5.99	4.87	2.46	3.33		
0	100	<i>Lipton</i>	without	4.49	41.3	0.77	3.16	26.9	13.9	1.02	18.8	2.81	5.73	4.54	2.51	3.29		
0	100	<i>Lipton</i>	without	4.23	44.7	0.51	3.04	28.1	14.4	0.97	18.2	3.31	5.74	4.99	2.50	2.99		
0	100	<i>Lipton</i>	with	—	45.8	0.86	3.24	28.7	15.0	1.00	16.8	3.08	5.59	5.03	2.51	2.61		
	MEAN			4.42	43.2	0.77	3.21	27.8	14.5	1.03	18.1	3.05	5.76	4.86	2.49	3.06		
0	200	<i>Bristol</i>	with	3.90	47.5	0.40	2.99	24.3	17.2	1.23	17.8	2.71	5.75	5.03	2.34	3.06		
0	200	<i>Bristol</i>	with	3.71	50.3	0.54	3.09	24.4	18.5	1.31	18.0	2.73	5.82	4.87	2.54	3.08		
0	200	<i>Bristol</i>	with	3.96	50.1	0.33	3.54	26.3	19.4	1.31	19.2	2.90	5.61	5.62	2.43	2.16		
0	200	<i>Bristol</i>	with	—	50.8	1.09	3.00	26.4	17.4	1.13	12.4	2.98	5.86	5.00	2.53	2.54		
	MEAN			4.05	49.7	0.59	3.16	25.4	18.1	1.25	16.9	2.83	5.76	5.13	2.46	2.71		
0	200	<i>Bristol</i>	without	4.06	48.7	0.92	2.87	28.6	17.8	1.18	16.3	3.15	5.74	5.02	2.34	2.90		
0	200	<i>Bristol</i>	without	4.52	40.3	0.50	2.90	24.1	15.5	1.08	1.81	3.24	6.14	5.12	2.49	2.88		
0	200	<i>Bristol</i>	without	3.14	51.1	0.88	2.91	27.1	19.6	1.10	16.2	3.11	5.99	5.44	2.47	2.65		
0	200	<i>Bristol</i>	with	—	49.4	0.49	2.63	24.7	16.1	1.02	12.7	3.13	5.91	5.31	2.51	2.50		
	MEAN			3.86	47.4	0.70	2.83	26.1	17.2	1.10	11.8	3.16	5.94	5.22	2.45	2.73		
0	200	<i>Lipton</i>	with	3.41	58.6	1.81	3.25	23.7	14.2	1.07	13.7	2.51	5.66	4.74	2.44	3.65		
0	200	<i>Lipton</i>	with	4.19	44.9	0.41	3.46	24.5	15.3	1.09	13.2	2.88	5.53	4.78	2.30	3.75		
0	200	<i>Lipton</i>	with	3.70	47.6	0.40	3.18	26.4	16.2	1.07	14.0	3.00	5.77	4.66	2.38	3.25		
0	200	<i>Lipton</i>	with	—	46.7	0.55	2.89	26.5	12.1	0.91	9.68	3.03	5.42	5.05	2.32	3.84		
	MEAN			3.97	49.4	0.79	3.20	25.3	14.4	1.04	12.6	2.86	5.60	4.81	2.36	3.62		

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -SP	K	Ca	Mg	Cl	S	P	Ca		Mg
2001/2002															
0	200	Lipton	with	47.6	0.56	3.42	29.4	15.9	1.09	17.9	3.14	5.57	4.94	2.43	4.03
0	200	Lipton	with	46.6	0.72	2.99	22.5	13.8	0.95	1.72	3.45	5.53	5.38	2.48	3.73
0	200	Lipton	with	45.7	0.62	3.07	29.3	15.4	1.04	13.7	3.13	5.64	4.70	2.45	3.95
0	200	Lipton	with	44.7	0.42	3.08	28.3	14.3	1.02	10.04	—	5.44	4.75	2.40	3.00
MEAN				46.1	0.58	3.14	27.4	14.8	1.03	11.0	3.19	5.55	4.94	2.44	3.68
100	100	Bristol	with	41.9	1.03	2.98	22.3	15.0	1.22	1.80	3.03	6.22	5.32	2.31	3.17
100	100	Bristol	with	43.2	1.23	2.92	25.0	13.7	1.13	1.34	3.28	6.33	5.90	2.55	2.63
100	100	Bristol	with	44.7	0.96	4.05	34.2	18.0	1.32	2.60	3.57	6.31	5.84	2.53	2.49
100	100	Bristol	with	41.9	1.25	3.20	25.3	13.4	1.18	13.20	—	5.86	5.39	2.88	2.58
MEAN				42.9	1.12	3.29	26.7	15.0	1.21	1.74	3.30	6.18	5.61	2.56	2.72
100	100	Bristol	with	43.5	0.70	3.33	27.7	16.7	1.24	2.61	3.23	6.13	5.14	2.48	2.43
100	100	Bristol	with	42.9	1.36	3.71	30.1	14.9	1.04	2.76	3.07	5.68	4.95	2.57	3.11
100	100	Bristol	with	43.1	1.34	2.70	25.0	15.3	0.96	2.78	3.61	6.12	5.23	2.73	2.53
100	100	Bristol	with	54.1	1.20	3.19	30.7	17.5	1.19	23.84	—	6.34	6.11	2.59	2.30
MEAN				45.9	1.15	3.23	28.4	16.1	1.11	2.67	3.44	6.07	5.36	2.59	2.59
100	100	Lipton	with	36.2	3.09	3.60	26.2	14.8	1.19	3.08	2.98	5.98	4.77	2.47	3.24
100	100	Lipton	with	40.5	1.45	2.97	23.1	12.9	0.95	1.09	3.16	5.79	4.83	2.56	2.45
100	100	Lipton	with	46.9	1.27	3.18	26.3	15.7	0.94	5.13	3.24	5.68	5.29	2.50	3.08
100	100	Lipton	with	47.6	0.77	2.93	26.7	13.7	0.95	13.29	—	6.09	4.92	2.57	3.86
MEAN				42.8	1.65	3.17	25.6	14.3	1.01	2.64	3.17	5.89	4.96	2.52	3.16
100	100	Lipton	with	38.2	1.26	2.99	22.8	12.0	0.96	2.42	3.21	5.86	4.58	2.49	3.78
100	100	Lipton	with	50.6	0.72	3.10	29.3	15.7	0.98	12.7	3.30	5.78	5.36	2.47	3.22
100	100	Lipton	with	44.7	1.41	3.05	24.4	14.5	0.92	3.23	3.30	5.76	4.93	2.76	3.19
100	200	Lipton	with	44.5	0.84	2.91	25.8	12.5	0.86	23.50	—	5.97	5.55	2.54	3.42
MEAN				44.5	1.06	3.01	25.6	13.7	0.93	5.12	3.33	5.84	5.10	2.57	3.40
100	200	Bristol	with	50.3	1.13	3.13	27.2	17.7	1.22	1.88	3.30	5.88	4.85	2.40	4.03
100	200	Bristol	with	47.9	2.82	3.00	23.8	16.9	1.18	1.62	3.47	6.06	5.15	2.44	2.99
100	200	Bristol	with	51.5	1.08	2.84	24.1	17.8	1.07	1.95	3.42	5.75	5.11	2.55	3.44
100	200	Bristol	with	53.4	2.11	3.25	30.5	19.0	1.29	23.69	—	6.23	5.11	2.49	3.07
MEAN				47.9	1.79	3.06	26.4	17.9	1.19	1.94	3.47	5.98	5.05	2.47	3.38

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
2001/2002																
100	200	<i>Bristol</i>	87thout	—	48.9	1.01	3.42	27.2	18.8	1.32	m.v.	2.87	5.72	5.07	2.42	2.94
100	200	<i>Bristol</i>	68thout	—	47.1	1.04	2.95	24.5	17.8	1.18	3.38	3.61	5.66	5.68	2.44	2.44
100	200	<i>Bristol</i>	82thout	—	46.2	0.98	3.50	32.7	20.7	1.33	4.33	3.82	6.10	5.35	2.48	2.83
100	100	<i>Bristol</i>	68thout	—	45.2	0.59	2.66	26.3	17.2	1.03	2.36	2.99	5.91	5.10	2.53	3.23
MEAN				7.66	46.8	0.91	3.13	27.7	18.6	1.22	3.36	3.32	5.85	5.30	2.47	2.86
100	200	<i>Lip800</i>	with	—	52.5	1.47	3.60	28.0	15.7	1.03	2.13	3.27	5.63	4.95	2.46	4.73
100	200	<i>Lip606</i>	with	—	44.2	0.71	3.15	24.8	13.7	0.93	1.56	3.45	5.89	4.34	2.62	4.23
100	200	<i>Lip881</i>	with	—	49.3	1.08	4.48	27.3	17.3	1.37	3.02	3.64	5.82	5.56	2.44	2.78
100	200	<i>Lip816</i>	with	—	48.8	1.14	3.37	25.6	13.8	1.16	2.30	3.11	5.75	4.98	2.55	3.73
MEAN				7.69	47.7	0.99	3.36	27.1	17.1	1.17	2.81	3.34	5.81	5.15	2.49	3.31
100	200	<i>Lipton</i>	67thout	—	46.1	1.09	3.52	23.2	15.5	1.14	4.26	3.52	5.65	4.83	2.45	3.33
100	200	<i>Lipton</i>	62thout	—	45.7	1.22	3.04	28.1	16.8	1.13	23.0	3.31	5.94	5.60	2.38	2.72
100	200	<i>Lipton</i>	85thout	—	49.4	1.28	4.24	31.9	17.7	1.20	4.44	3.56	5.63	5.08	2.46	3.49
100	200	<i>Lipton</i>	92thout	—	50.8	0.88	4.27	36.1	18.1	1.21	2.11	3.49	5.63	5.09	2.64	3.40
MEAN				7.71	48.0	1.12	3.77	29.8	17.0	1.17	8.45	3.47	5.71	5.15	2.48	3.24
2002/2003																
0	100	<i>Bris63</i>	with	—	36.4	1.95	3.35	12.5	19.9	1.76	5.23	2.81	6.62	4.36	2.85	2.82
0	100	<i>Bris62</i>	with	—	36.5	1.77	3.38	13.3	20.0	1.80	0.57	2.84	6.65	4.78	2.83	3.35
0	100	<i>Bris67</i>	with	—	35.8	3.20	3.79	13.5	20.5	2.01	2.94	2.97	7.02	4.16	2.90	3.15
0	100	<i>Bris69</i>	with	—	34.9	3.14	3.00	13.4	18.0	1.67	0.56	2.88	7.02	4.60	2.93	3.06
MEAN				5.25	35.9	2.52	3.38	13.2	19.6	1.81	2.33	2.87	6.83	4.48	2.88	3.10
0	100	<i>Bristol</i>	55thout	—	36.0	n.a.	3.63	14.5	18.5	1.74	1.38	2.57	6.18	3.98	2.68	2.64
0	100	<i>Bristol</i>	52thout	—	39.0	2.13	3.07	14.2	18.0	1.67	6.43	2.88	6.73	4.33	2.83	3.03
0	100	<i>Bristol</i>	57thout	—	37.4	3.05	3.20	18.1	17.3	1.66	7.69	3.26	6.39	4.52	3.06	3.32
0	100	<i>Bristol</i>	56thout	—	37.1	2.67	3.67	17.0	19.3	2.03	6.21	2.95	6.59	4.59	2.90	2.99
MEAN				5.54	37.4	2.62	3.39	15.9	18.3	1.78	5.43	2.92	6.47	4.36	2.87	3.00

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
mg g ⁻¹																
2002/2003																
0	100	<i>Lipton</i>	with	4.42	35.1	1.75	3.59	15.0	18.0	1.74	3.81	2.82	6.78	5.36	2.97	2.83
0	100	<i>Lipton</i>	with	4.80	38.3	1.82	3.69	14.3	16.7	1.57	0.80	2.70	6.60	4.35	2.87	2.65
0	100	<i>Lipton</i>	with	4.65	35.7	1.57	3.02	15.7	17.5	1.57	1.57	2.86	6.85	4.12	2.96	3.51
0	100	<i>Lipton</i>	with	—	36.5	n.a.	3.66	15.2	18.6	1.86	3.74	2.85	6.23	4.32	2.89	0.9
	MEAN			4.83	36.4	1.71	3.49	15.0	17.7	1.69	2.48	2.81	6.62	4.53	2.92	3.02
0	100	<i>Lipton</i>	without	4.72	36.8	n.a.	2.97	12.3	18.2	1.61	1.13	2.69	6.30	4.35	2.93	3.01
0	100	<i>Lipton</i>	without	4.96	38.4	1.83	4.13	13.1	15.9	1.80	0.64	2.85	6.43	4.40	2.88	3.01
0	100	<i>Lipton</i>	without	5.04	36.6	2.24	3.18	12.2	20.0	2.02	0.52	2.85	6.67	4.32	2.97	3.30
0	100	<i>Lipton</i>	without	—	36.1	n.a.	4.02	14.8	16.3	1.79	0.66	3.18	6.35	4.64	3.02	0.87
	MEAN			4.92	37.0	2.04	3.58	13.1	17.6	1.81	0.74	2.89	6.44	4.43	2.95	3.05
0	200	<i>Bristol</i>	with	4.55	48.0	1.42	3.50	14.7	14.0	1.40	1.34	2.69	6.28	3.89	2.81	3.87
0	200	<i>Bristol</i>	with	4.43	57.7	1.29	3.71	16.3	15.1	1.61	4.63	2.76	6.46	4.46	2.91	4.00
0	200	<i>Bristol</i>	with	5.18	50.4	1.92	3.06	18.7	16.9	1.43	6.93	3.02	6.52	4.27	2.94	3.93
0	200	<i>Bristol</i>	with	—	44.1	1.30	3.22	19.3	16.3	1.59	7.11	2.74	6.20	4.49	2.88	0.52
	MEAN			4.75	50.1	1.48	3.37	17.2	15.5	1.51	5.00	2.80	6.36	4.28	2.87	4.08
0	200	<i>Bristol</i>	without	4.32	46.0	1.06	3.97	15.0	15.9	1.61	1.12	2.91	6.00	4.36	2.85	3.64
0	200	<i>Bristol</i>	without	4.53	46.8	1.49	3.56	12.5	17.5	1.56	3.74	2.90	6.17	4.28	2.90	3.88
0	200	<i>Bristol</i>	without	5.11	48.5	1.90	3.73	11.5	20.4	1.92	3.12	3.01	5.81	4.33	2.86	4.33
0	200	<i>Bristol</i>	without	—	47.5	1.80	3.06	17.2	17.4	1.59	8.67	3.07	5.88	4.22	2.95	0.97
	MEAN			4.88	47.2	1.56	3.58	14.1	17.8	1.67	4.16	2.97	5.97	4.30	2.89	3.96
0	200	<i>Lipton</i>	with	3.78	48.4	0.62	3.17	15.6	17.5	1.53	1.64	2.86	6.18	4.15	2.77	3.98
0	200	<i>Lipton</i>	with	4.04	44.9	1.03	3.26	13.2	15.1	1.48	1.18	3.03	6.10	4.25	2.92	3.69
0	200	<i>Lipton</i>	with	4.50	48.8	0.99	3.24	12.3	15.7	1.45	0.90	3.02	6.49	4.12	2.96	4.19
0	200	<i>Lipton</i>	with	—	47.2	n.a.	3.10	15.7	14.6	1.36	1.49	2.95	6.31	4.55	2.94	0.42
	MEAN			4.29	47.3	0.88	3.19	14.2	15.7	1.46	1.31	2.96	6.27	4.27	2.89	4.07

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca		Mg
mg g ⁻¹																
2002/2003																
0	200	<i>Lipton</i>	without	4.14	43.4	1.12	3.22	19.6	17.9	1.55	8.71	3.02	5.98	4.01	2.91	3.70
0	200	<i>Lipton</i>	without	4.22	48.4	0.94	3.22	15.4	17.4	1.66	1.22	2.90	6.00	4.28	2.73	4.51
0	200	<i>Lipton</i>	without	4.64	49.1	2.02	3.83	13.2	17.3	1.67	0.62	3.02	6.29	4.04	2.91	4.32
0	200	<i>Lipton</i>	with	—	48.2	1.64	3.11	15.5	16.6	1.76	1.02	3.01	6.19	4.12	2.92	3.28
MEAN				4.48	47.3	1.43	3.35	15.9	17.3	1.66	2.89	2.99	6.12	4.11	2.87	3.96
100	100	<i>Bristol</i>	with	6.49	37.3	3.55	3.85	17.2	14.4	1.49	4.20	2.99	6.65	4.14	2.83	3.33
100	100	<i>Bristol</i>	with	6.07	36.1	3.46	3.55	12.2	17.2	1.46	3.46	2.90	6.56	4.34	2.89	2.59
100	100	<i>Bristol</i>	with	6.20	36.1	4.90	4.25	20.2	14.4	1.63	4.69	3.54	6.14	5.14	3.09	3.41
100	100	<i>Bristol</i>	with	—	33.7	3.49	3.46	12.8	16.8	1.57	4.18	3.14	6.61	4.45	2.94	3.30
MEAN				6.34	35.8	3.85	3.78	15.6	15.7	1.54	4.13	3.14	6.49	4.52	2.94	3.16
100	100	<i>Bristol</i>	without	6.87	37.9	n.a.	3.05	12.7	18.1	1.54	5.49	2.97	6.32	4.54	2.86	2.48
100	100	<i>Bristol</i>	without	6.80	37.8	3.38	3.61	15.5	14.7	1.48	4.87	3.20	6.54	4.35	2.86	2.88
100	100	<i>Bristol</i>	without	6.04	36.3	3.44	2.97	14.1	18.2	1.64	5.68	3.07	6.62	4.44	2.89	3.38
100	100	<i>Bristol</i>	with	—	36.7	3.61	3.53	13.1	17.1	1.67	3.14	2.92	6.64	4.51	2.94	2.65
MEAN				6.64	37.2	3.48	3.29	13.9	17.0	1.58	4.80	3.04	6.53	4.46	2.89	2.85
100	100	<i>Lipton</i>	with	—	34.3	2.78	3.96	15.0	16.5	1.67	0.91	2.83	6.56	4.45	2.84	2.95
100	100	<i>Lipton</i>	with	—	37.2	3.11	3.55	15.5	19.4	1.87	1.10	3.03	6.43	4.49	2.92	2.75
100	100	<i>Lipton</i>	with	—	35.3	2.72	4.30	15.4	16.4	1.70	1.35	2.99	6.65	5.58	3.04	3.33
100	100	<i>Lipton</i>	with	—	36.7	3.10	3.88	17.0	16.1	1.65	1.32	2.96	6.55	4.40	2.99	3.37
MEAN				5.49	35.8	2.93	3.92	15.7	17.1	1.72	1.17	2.96	6.55	4.73	2.95	3.10
100	100	<i>Lipton</i>	with	—	33.8	3.89	3.48	17.6	15.5	1.39	6.16	2.74	6.35	4.32	2.92	2.71
100	100	<i>Lipton</i>	with	—	36.5	2.23	3.58	19.2	17.9	1.93	8.84	3.18	6.45	4.34	2.93	3.08
100	100	<i>Lipton</i>	with	—	36.1	n.a.	3.78	17.1	15.8	1.64	5.09	2.86	6.55	4.40	2.95	3.59
100	100	<i>Lipton</i>	with	—	34.2	3.12	3.83	21.0	15.0	1.60	7.63	2.78	6.39	4.48	2.86	2.83
MEAN				5.50	35.2	3.08	3.67	18.7	16.0	1.64	6.93	2.89	6.43	4.38	2.91	3.05

Table A.10: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds				Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	
2002/2003															
100	200	<i>Brisol</i>	with	46.4	n.a.	3.60	14.3	19.3	1.91	0.94	3.40	5.95	4.48	2.90	4.08
100	200	<i>Brisol</i>	with	47.2	2.68	3.09	16.4	18.3	1.62	7.62	3.31	6.08	4.37	2.84	3.98
100	200	<i>Brisol</i>	with	46.0	3.80	3.35	14.3	19.0	1.69	0.92	2.94	6.09	4.08	2.91	3.93
100	200	<i>Brisol</i>	with	50.7	2.70	3.74	14.3	20.9	2.13	0.56	3.42	6.70	4.34	3.14	4.35
MEAN				47.6	3.06	3.45	14.8	19.4	1.84	2.51	3.27	6.21	4.32	2.95	4.08
100	200	<i>Bristol</i>	with	48.0	2.51	3.25	14.0	14.2	1.28	1.77	3.31	5.83	4.53	2.91	3.38
100	200	<i>Bristol</i>	with	49.2	2.22	3.14	15.9	17.8	1.70	1.40	3.22	6.16	4.20	2.85	3.57
100	200	<i>Bristol</i>	with	49.4	2.58	3.64	15.6	19.9	2.02	1.10	3.67	6.27	4.21	2.93	3.95
100	200	<i>Bristol</i>	with	47.4	3.37	3.30	16.6	19.0	1.78	0.92	3.38	6.09	4.50	2.95	3.98
MEAN				48.5	2.67	3.33	15.5	17.7	1.70	1.30	3.40	6.09	4.36	2.91	3.72
100	200	<i>Lipton</i>	with	48.9	2.30	3.93	12.5	16.1	1.57	2.68	3.20	6.25	3.92	2.86	3.92
100	200	<i>Lipton</i>	with	49.4	1.99	3.13	18.6	17.0	1.54	6.54	3.18	6.14	4.69	2.88	4.44
100	200	<i>Lipton</i>	with	49.4	1.98	3.40	10.7	19.0	1.78	2.16	3.31	6.15	4.00	2.95	4.33
100	200	<i>Lipton</i>	with	46.2	2.24	3.88	15.6	15.0	1.71	4.31	3.13	6.30	4.45	2.85	4.47
MEAN				48.5	2.13	3.59	14.3	16.8	1.65	3.92	3.20	6.21	4.27	2.89	4.29
100	200	<i>Lipton</i>	with	46.1	2.33	3.51	15.3	15.3	1.38	7.05	3.43	6.04	4.24	2.93	3.90
100	200	<i>Lipton</i>	with	51.7	2.11	3.22	15.6	13.5	1.28	1.28	3.24	6.15	4.03	2.91	3.93
100	200	<i>Lipton</i>	with	48.8	2.63	3.34	15.5	15.3	1.41	1.37	3.60	6.04	4.24	2.98	4.24
100	200	<i>Lipton</i>	with	41.2	2.76	2.97	13.5	14.3	1.42	0.83	3.13	6.27	4.24	2.94	3.85
MEAN				46.9	2.46	3.26	14.9	14.6	1.37	2.63	3.35	6.13	4.19	2.94	3.98

note: n.a. – not available

Table A.11: Mineral nutrient content in leaf and seed samples and the recorded seed yield, Inverness, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds						Yield t ha ⁻¹			
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg				
														mg g ⁻¹					
														2000/2001					
0	100	<i>Bristol</i>	with	n.a.	n.a.	1.17	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.55	5.65	5.59	2.39	2.68	
0	100	<i>Bristol</i> 198	with	48.4	48.4	0.73	2.29	31.8	14.6	1.07	13.6	n.a.	2.10	5.81	4.24	2.67	1.80		
0	100	<i>Bristol</i> 113	with	48.5	48.5	0.81	2.25	32.8	16.5	0.91	15.3	n.a.	2.08	5.21	4.83	2.21	2.38		
0	100	<i>Bristol</i> 162	with	50.5	50.5	0.58	2.16	31.2	15.7	0.89	14.05	n.a.	—	5.50	4.49	2.51	2.10		
MEAN				49.1	49.1	0.82	2.23	31.9	15.6	0.96	14.5	n.a.	2.20	5.54	4.79	2.45	2.24		
0	100	<i>Bristol</i>	without	49.1	49.1	0.72	2.46	31.5	16.3	0.91	15.9	n.a.	2.14	5.54	4.91	2.33	1.60		
0	100	<i>Bristol</i>	without	49.8	49.8	0.50	2.41	33.5	16.8	0.94	15.3	n.a.	2.42	5.44	5.04	2.38	2.27		
0	100	<i>Bristol</i>	without	49.2	49.2	0.64	2.19	30.2	14.5	0.78	12.7	n.a.	2.33	5.48	5.12	2.24	2.25		
0	100	<i>Bristol</i>	without	53.1	53.1	1.04	3.05	33.9	16.6	0.99	13.42	n.a.	—	5.35	5.30	2.34	2.59		
MEAN				50.3	50.3	0.72	2.53	32.3	16.1	0.91	14.5	n.a.	2.33	5.45	5.09	2.32	2.18		
0	100	<i>Lipton</i>	with	n.a.	n.a.	0.84	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.20	5.31	5.08	2.41	2.38		
0	100	<i>Lipton</i>	with	n.a.	n.a.	0.52	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.40	5.66	4.12	2.69	2.17		
0	100	<i>Lipton</i> 159	with	49.9	49.9	0.79	2.74	29.3	14.0	0.89	9.13	n.a.	2.07	5.47	4.61	2.39	2.62		
0	100	<i>Lipton</i> 163	with	50.3	50.3	0.87	2.78	31.9	15.1	1.04	12.12	n.a.	—	5.79	4.55	2.66	2.01		
MEAN				50.1	50.1	0.75	2.76	30.6	14.5	0.97	10.6	n.a.	2.20	5.56	4.59	2.54	2.30		
0	100	<i>Lipton</i>	without	n.a.	n.a.	1.01	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.21	5.66	4.96	2.59	1.80		
0	100	<i>Lipton</i>	without	n.a.	n.a.	1.01	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.44	5.63	4.76	2.45	2.02		
0	100	<i>Lipton</i>	without	46.2	46.2	0.85	2.56	31.5	18.0	0.99	17.3	n.a.	2.37	5.51	4.88	2.31	2.31		
0	100	<i>Lipton</i>	without	49.1	49.1	1.07	2.64	29.5	15.2	0.87	11.86	n.a.	—	5.60	4.87	2.41	2.64		
MEAN				47.6	47.6	0.98	2.60	30.5	16.6	0.93	14.6	n.a.	2.37	5.60	4.87	2.44	2.20		
0	200	<i>Bristol</i> 173	with	52.7	52.7	0.77	2.93	29.5	15.2	0.99	9.73	n.a.	2.09	4.85	5.02	2.29	3.13		
0	200	<i>Bristol</i> 194	with	51.1	51.1	0.90	2.19	28.0	15.7	1.07	11.5	n.a.	2.30	5.12	4.57	2.53	2.91		
0	200	<i>Bristol</i> 128	with	53.7	53.7	0.86	2.18	29.1	15.2	0.84	10.4	n.a.	2.13	5.06	4.85	2.35	2.65		
0	200	<i>Bristol</i>	with	n.a.	n.a.	0.63	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	—	5.46	4.51	2.68	2.78		
MEAN				52.5	52.5	0.79	2.43	28.9	15.4	0.97	10.5	n.a.	2.16	5.12	4.74	2.46	2.87		

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
mg g ⁻¹																
2000/2001																
0	200	<i>Bristol</i>	without	3.31	54.7	0.76	2.59	31.6	16.0	0.94	12.5	2.11	5.73	5.00	2.58	1.53
0	200	<i>Bristol</i>	without	n.a.	n.a.	0.75	n.a.	n.a.	n.a.	n.a.	n.a.	2.19	5.49	4.47	2.71	2.28
0	200	<i>Bristol</i>	with		54.2	0.59	2.83	30.0	16.9	1.03	11.2	2.37	5.20	4.93	2.26	2.29
0	200	<i>Bristol</i>	with		55.2	0.97	2.54	30.0	16.2	0.95	12.43		5.36	4.71	2.47	2.29
MEAN				3.75	54.7	0.77	2.65	30.5	16.4	0.97	11.6	2.28	5.44	4.78	2.50	2.19
0	200	<i>Lipton</i>	58 with		51.0	0.74	2.43	28.2	16.2	0.94	10.7	2.29	5.01	4.72	2.29	3.06
0	200	<i>Lipton</i>	88 with		49.8	0.73	2.60	30.0	18.0	1.19	3.29	2.49	5.39	4.10	2.70	2.57
0	200	<i>Lipton</i>	with	n.a.	n.a.	1.12	n.a.	n.a.	n.a.	n.a.	n.a.	2.29	5.22	4.56	2.37	2.72
0	200	<i>Lipton</i>	91 with		51.0	0.60	2.34	26.5	14.6	0.85	9.22		5.68	4.51	2.70	2.72
MEAN				4.79	50.6	0.80	2.46	28.2	16.3	0.99	7.75	2.33	5.32	4.47	2.52	2.84
0	200	<i>Lipton</i>	without	n.a.	n.a.	0.86	n.a.	n.a.	n.a.	n.a.	n.a.	2.42	5.77	4.96	2.47	1.79
0	200	<i>Lipton</i>	without	n.a.	n.a.	0.87	n.a.	n.a.	n.a.	n.a.	n.a.	2.44	5.13	4.53	2.50	2.80
0	200	<i>Lipton</i>	with		50.6	0.67	2.92	32.5	21.4	1.12	15.1	2.59	5.22	4.82	2.39	2.54
0	200	<i>Lipton</i>	with		49.2	0.41	2.50	28.3	17.1	0.87	12.34		5.22	4.82	2.39	2.54
MEAN				3.81	49.9	0.70	2.71	30.4	19.3	1.00	13.6	2.45	5.33	4.78	2.43	2.51
100	100	<i>Bristol</i>	64 with		48.9	5.21	2.28	28.0	17.6	0.88	4.37	2.71	5.74	5.36	2.57	2.89
100	100	<i>Bristol</i>	with	n.a.	n.a.	m.v.	n.a.	n.a.	n.a.	n.a.	n.a.	2.45	5.47	3.97	2.76	2.26
100	100	<i>Bristol</i>	with	n.a.	n.a.	5.68	n.a.	n.a.	n.a.	n.a.	n.a.	2.60	5.21	4.86	2.35	2.89
100	100	<i>Bristol</i>	with	n.a.	n.a.	4.92	n.a.	n.a.	n.a.	n.a.	12.63		5.78	4.64	2.69	2.89
MEAN				6.64	48.9	5.27	2.28	28.0	17.6	0.88	4.37	2.60	5.55	4.71	2.59	2.69
100	100	<i>Bristol</i>	with		52.7	4.12	2.44	29.6	16.8	0.84	3.51	2.64	5.51	4.83	2.38	2.00
100	100	<i>Bristol</i>	without	n.a.	n.a.	2.41	n.a.	n.a.	n.a.	n.a.	n.a.	2.82	5.57	5.07	2.40	2.23
100	100	<i>Bristol</i>	with		51.9	2.33	2.16	27.1	18.2	0.82	4.58	2.80	5.46	5.27	2.23	2.62
100	100	<i>Bristol</i>	with		54.8	4.96	2.98	29.9	16.7	0.93	3.27		5.36	5.05	2.24	2.62
MEAN				7.13	53.1	3.46	2.53	28.9	17.3	0.86	3.78	2.75	5.47	5.06	2.31	2.34

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹			
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg	
2000/2001																	
100	100	Lipton	with	n.a.	n.a.	3.56	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.94	5.75	5.44	2.53	2.59
100	100	Lipton	with	n.a.	n.a.	1.20	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.37	5.72	3.81	2.67	2.35
100	100	Lipton	with	n.a.	n.a.	3.30	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.61	5.48	4.79	2.31	2.75
100	100	Lipton	with	n.a.	51.8	3.96	3.06	30.5	15.8	1.16	3.65	2.71	5.53	4.62	2.71	1.98	1.98
MEAN				7.40	51.8	3.00	3.06	30.5	15.8	1.16	3.66	2.55	2.62	5.62	4.67	2.55	2.42
100	100	Lipton	without	n.a.	50.9	4.53	3.23	29.6	15.8	0.99	3.39	2.46	2.70	5.50	4.96	2.46	1.94
100	100	Lipton	without	n.a.	n.a.	1.17	n.a.	n.a.	n.a.	n.a.	n.a.	2.43	2.73	5.60	4.91	2.43	2.23
100	100	Lipton	without	n.a.	n.a.	1.02	n.a.	n.a.	n.a.	n.a.	n.a.	2.44	2.84	5.50	5.03	2.44	2.46
100	100	Lipton	without	n.a.	n.a.	3.27	n.a.	n.a.	n.a.	n.a.	2.80	2.48	2.79	5.60	5.11	2.48	2.64
MEAN				7.21	50.9	2.50	3.23	29.6	15.8	0.99	3.39	2.45	2.79	5.55	5.00	2.45	2.32
100	200	Bristol	with	n.a.	53.9	5.16	2.15	25.6	19.4	0.86	3.90	2.55	3.30	5.56	5.05	2.55	3.35
100	200	Bristol	with	n.a.	57.2	1.26	2.53	28.8	16.1	1.15	11.5	2.51	3.40	4.82	4.79	2.51	3.80
100	200	Bristol	with	n.a.	n.a.	5.63	n.a.	n.a.	n.a.	n.a.	n.a.	2.37	3.07	4.76	4.59	2.37	3.62
100	200	Bristol	with	n.a.	56.0	4.70	2.80	30.0	19.4	1.14	3.80	2.63	3.20	5.39	4.56	2.63	3.82
MEAN				5.72	55.7	4.19	2.49	28.1	18.3	1.05	6.41	2.51	3.20	5.13	4.75	2.51	3.64
100	200	Bristol	without	n.a.	54.4	4.12	2.54	27.3	18.4	1.01	4.02	2.39	2.99	5.19	4.75	2.39	2.84
100	200	Bristol	without	n.a.	56.0	2.44	2.65	28.2	16.3	1.35	3.11	2.85	2.93	5.44	4.15	2.85	2.92
100	200	Bristol	without	n.a.	53.3	2.43	2.14	25.8	19.8	0.91	4.70	2.33	3.25	5.05	4.81	2.33	3.37
100	200	Bristol	without	n.a.	58.0	5.19	2.40	24.8	14.8	0.79	23.1	2.29	3.31	4.80	4.74	2.29	3.31
MEAN				6.87	55.4	3.54	2.43	26.5	17.3	1.02	3.49	2.47	3.07	5.12	4.61	2.47	3.11
100	200	Lipton	with	n.a.	n.a.	3.23	n.a.	n.a.	n.a.	n.a.	n.a.	2.48	2.97	5.25	4.61	2.48	3.56
100	200	Lipton	with	n.a.	n.a.	1.47	n.a.	n.a.	n.a.	n.a.	n.a.	2.66	2.91	5.22	4.04	2.66	2.81
100	200	Lipton	with	n.a.	52.5	4.65	2.28	25.7	16.3	0.87	3.37	2.38	2.89	5.12	4.74	2.38	3.58
100	200	Lipton	with	n.a.	n.a.	3.47	n.a.	n.a.	n.a.	n.a.	2.81	2.64	3.18	5.24	4.45	2.64	3.18
MEAN				6.00	52.5	3.20	2.28	25.7	16.3	0.87	3.37	2.54	2.90	5.21	4.46	2.54	3.28

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
2000/2001																
100	200	<i>Lipton</i>	without	n.a.	n.a.	4.75	n.a.	n.a.	n.a.	n.a.	n.a.	3.05	4.93	4.29	2.42	2.84
100	200	<i>Lipton</i>	without	n.a.	n.a.	0.92	n.a.	n.a.	n.a.	n.a.	n.a.	2.93	5.51	4.62	2.57	3.05
100	200	<i>Lipton</i>	without	n.a.	n.a.	1.67	n.a.	n.a.	n.a.	n.a.	n.a.	3.03	5.30	4.72	2.56	3.38
100	200	<i>Lipton</i>	without	n.a.	n.a.	4.31	n.a.	n.a.	n.a.	n.a.	n.a.	—	5.37	4.69	2.45	3.17
MEAN				n.a.	n.a.	2.91	n.a.	n.a.	n.a.	n.a.	n.a.	3.06	5.28	4.58	2.50	3.11
2001/2002																
0	100	<i>Bristol</i>	with	3.59	45.4	0.32	3.58	21.1	15.8	1.45	7.13	2.40	5.68	5.17	2.70	2.52
0	100	<i>Bristol</i>	with	—	50.1	0.12	3.99	21.8	22.3	1.61	8.30	2.10	5.64	4.43	2.55	2.05
0	100	<i>Bristol</i>	with	—	42.6	0.38	3.16	14.8	18.9	1.37	3.89	2.49	5.46	4.75	2.61	2.31
0	100	<i>Bristol</i>	with	—	42.9	0.31	3.16	14.3	18.5	1.21	6.08	—	5.77	4.42	2.49	2.26
MEAN				3.25	45.2	0.28	3.47	18.0	18.9	1.41	6.35	2.32	5.64	4.69	2.59	2.29
0	100	<i>Bristol</i>	without	—	50.5	0.49	3.75	16.5	21.5	1.76	4.74	2.38	5.71	5.21	2.68	1.93
0	100	<i>Bristol</i>	without	—	49.3	0.21	3.86	19.3	21.7	1.66	5.35	2.29	5.29	4.58	2.65	2.04
0	100	<i>Bristol</i>	without	—	42.5	0.54	3.42	18.1	19.5	1.33	5.73	2.32	5.44	4.22	2.50	2.09
0	100	<i>Bristol</i>	without	—	48.8	0.19	3.51	16.2	20.3	1.50	4.08	—	5.59	4.70	2.60	2.03
MEAN				3.68	47.8	0.36	3.64	17.5	20.7	1.56	4.97	2.39	5.51	4.68	2.61	2.02
0	100	<i>Lipton</i>	with	—	43.0	0.37	4.34	19.0	21.5	1.62	4.94	2.02	5.14	4.75	2.70	2.41
0	100	<i>Lipton</i>	with	—	48.0	0.32	3.99	20.9	17.2	1.19	6.08	2.19	5.71	4.32	2.55	2.16
0	100	<i>Lipton</i>	with	—	45.9	0.44	3.36	15.5	18.4	1.21	3.98	2.32	5.46	4.38	2.57	2.48
0	100	<i>Lipton</i>	with	—	41.0	0.27	3.25	23.3	13.8	1.00	7.29	—	5.49	3.88	2.55	2.08
MEAN				3.51	44.4	0.35	3.74	19.7	17.7	1.26	5.73	2.18	5.45	4.33	2.59	2.28
0	100	<i>Lipton</i>	without	—	46.1	0.38	4.02	18.1	18.2	1.43	4.08	2.30	5.46	4.66	2.44	1.46
0	100	<i>Lipton</i>	without	—	49.6	0.35	4.02	17.6	19.3	1.51	4.64	2.40	5.12	4.26	2.69	2.20
0	100	<i>Lipton</i>	without	—	42.9	0.35	3.38	20.7	15.7	1.17	5.41	2.24	5.36	4.35	2.53	1.83
0	100	<i>Lipton</i>	without	—	40.5	0.30	3.56	16.7	17.6	1.29	5.24	—	5.56	4.33	2.60	1.80
MEAN				3.28	44.8	0.35	3.75	18.3	17.7	1.35	4.89	2.30	5.38	4.40	2.56	1.82

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹		
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca	Mg
2001/2002																
0	200	Bristol	with	3.39	54.7	0.11	3.79	20.5	22.9	1.59	4.90	2.28	5.61	4.20	2.59	2.44
0	200	Bristol	32 with		53.7	0.13	3.63	18.5	23.8	1.80	5.36	2.15	6.01	4.22	2.57	1.98
0	200	Bristol	36 with		48.3	0.26	3.50	15.7	19.3	1.61	2.39	2.39	5.40	4.57	2.59	2.69
0	200	Bristol	94 with		49.8	0.25	2.96	16.6	17.5	1.39	41795		5.22	3.91	2.6033	
	MEAN			3.25	51.7	0.19	3.47	17.8	20.9	1.60	4.36	2.19	5.56	4.22	2.59	2.36
0	200	Bristol	with		53.8	0.33	3.75	15.7	24.6	1.81	3.10	2.22	5.22	4.52	2.84	2.72
0	200	Bristol	with		50.9	0.24	3.86	20.9	24.9	1.84	7.62	2.40	4.94	4.55	2.90	2.29
0	200	Bristol	with		48.9	0.40	3.47	16.8	20.5	1.62	3.27	2.11	5.41	4.63	2.54	2.36
0	200	Bristol	with		49.7	0.33	3.30	18.8	21.2	1.57	52380		5.44	3.90	2.6517	
	MEAN			3.58	50.8	0.33	3.60	18.1	22.8	1.71	4.84	2.21	5.25	4.40	2.73	2.39
0	200	Lipton	08 with		51.7	0.18	3.54	17.8	16.2	1.16	2.83	2.17	5.69	4.04	2.68	3.19
0	200	Lipton	49 with		49.0	0.19	4.18	22.9	19.9	1.41	3.62	2.11	5.99	3.98	2.55	3.25
0	200	Lipton	40 with		52.5	0.34	4.04	18.1	19.6	1.54	3.42	2.65	5.32	4.12	2.79	2.61
0	200	Lipton	45 with		49.8	0.15	3.92	19.4	17.0	1.42	32102		5.42	3.86	2.5659	
	MEAN			3.36	50.8	0.22	3.92	19.5	18.2	1.38	3.24	2.26	5.60	4.00	2.64	2.91
0	200	Lipton	with		53.5	0.20	4.10	19.0	20.3	1.46	2.56	2.39	5.52	4.87	2.81	2.57
0	200	Lipton	with		53.4	0.40	4.44	20.4	19.5	1.50	2.57	2.51	5.10	4.11	2.92	2.82
0	200	Lipton	with		48.5	0.34	3.68	21.4	19.1	1.43	7.06	2.26	5.60	4.90	2.60	1.59
0	200	Lipton	with		46.2	0.21	3.40	15.8	17.3	1.32	32367		5.28	4.06	2.6645	
	MEAN			3.79	50.4	0.29	3.91	19.1	19.1	1.43	3.88	2.43	5.38	4.49	2.75	2.36
100	100	Bristol	85 with		44.5	2.00	3.25	22.9	16.1	1.29	1.65	2.70	5.63	4.21	2.66	2.33
100	100	Bristol	72 with		47.6	0.78	3.58	18.0	22.4	1.81	1.09	2.69	5.78	4.31	2.59	2.26
100	100	Bristol	47 with		44.8	3.94	3.10	15.4	20.9	1.61	0.63	3.01	5.76	3.94	2.64	2.22
100	100	Bristol	31 with		42.4	1.91	3.17	18.3	18.7	1.18	12786		5.85	4.42	2.6517	
	MEAN			7.34	44.8	2.16	3.28	18.7	19.5	1.47	1.28	2.79	5.75	4.22	2.64	2.25
100	100	Bristol	with		49.1	1.63	3.83	21.0	22.3	1.83	1.67	2.87	5.68	4.26	2.63	1.96
100	100	Bristol	with		49.3	1.06	3.80	20.8	24.9	1.74	2.01	2.83	5.48	4.10	2.70	2.15
100	100	Bristol	with		47.1	1.22	2.98	20.1	18.5	1.26	3.02	2.99	5.59	4.50	2.66	1.78
100	100	Bristol	with		46.8	0.76	3.59	17.2	20.2	1.63	12689		5.95	4.44	2.6372	
	MEAN			7.72	48.1	1.17	3.55	19.8	21.5	1.62	2.08	2.89	5.67	4.33	2.66	1.90

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds					Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg	
2001/2002																
100	100	Lipton	with	7.68	46.0	0.75	3.69	19.8	16.5	1.33	1.16	2.57	5.58	4.61	2.80	2.71
100	100	Lipton	35 with		44.7	1.52	4.10	16.5	18.7	1.48	0.51	2.68	5.57	5.01	2.61	2.63
100	100	Lipton	52 with		43.5	1.31	3.51	15.4	18.8	1.50	1.18	2.72	5.54	4.64	2.66	2.49
100	100	Lipton	26 with		44.4	1.24	3.51	18.7	18.4	1.24	1.20		5.73	4.40	2.62	2.48
MEAN				7.20	44.7	1.21	3.70	17.6	18.1	1.39	1.01	2.65	5.61	4.66	2.67	2.58
100	100	Lipton	with		46.9	0.92	4.00	19.4	20.0	1.39	1.68	2.61	5.89	5.38	2.71	2.01
100	100	Lipton	with		52.0	0.98	3.77	16.7	20.3	1.51	1.15	2.76	5.25	4.27	2.68	2.62
100	100	Lipton	with		45.7	1.41	3.50	17.8	15.7	1.29	0.95	2.75	5.67	4.28	2.59	1.80
100	100	Lipton	with		44.6	0.92	3.50	16.0	19.4	1.53	1.20		5.57	4.43	2.66	2.69
MEAN				7.76	47.3	1.06	3.69	17.5	18.8	1.43	1.26	2.76	5.59	4.59	2.66	2.09
100	200	Bristol	34 with		57.5	1.45	3.52	20.2	23.1	1.95	1.12	3.33	5.06	4.35	2.66	3.22
100	200	Bristol	84 with		39.9	1.21	3.41	18.7	25.0	1.92	1.73	3.43	5.34	4.46	2.66	2.49
100	200	Bristol	49 with		52.3	1.20	4.23	19.3	25.2	2.20	0.83	3.69	5.16	4.09	2.69	2.32
100	200	Bristol	with		m.v.	1.35	4.04	22.4	24.3	2.03	1.30		5.31	4.49	2.70	2.33
MEAN				8.22	51.1	1.30	3.80	20.2	24.4	2.03	1.19	3.43	5.22	4.35	2.68	2.54
100	200	Bristol	with		53.2	0.99	3.32	15.4	23.0	1.79	1.14	3.49	5.00	5.04	2.76	2.98
100	200	Bristol	with		45.3	1.01	3.90	19.3	25.9	1.82	0.99	3.46	5.11	4.53	2.86	2.20
100	200	Bristol	with		53.3	1.19	3.47	21.3	18.1	1.45	2.47	3.42	5.00	4.13	2.73	1.87
100	200	Bristol	with		51.7	1.06	3.69	17.6	21.8	1.80	1.30		5.21	4.34	2.70	2.36
MEAN				7.10	50.9	1.06	3.60	18.4	22.2	1.72	1.42	3.47	5.08	4.51	2.77	2.28
100	200	Lipton	49 with		54.0	2.55	3.69	16.4	19.7	1.49	0.61	3.23	5.28	4.67	2.71	3.06
100	200	Lipton	84 with		68.1	1.31	3.93	17.6	19.7	1.70	1.04	3.14	5.16	4.12	2.66	2.84
100	200	Lipton	02 with		51.2	1.19	3.76	19.4	19.8	1.52	0.92	3.46	5.22	4.36	2.73	2.95
100	200	Lipton	47 with		54.2	1.22	4.23	17.1	20.4	1.72	0.72		5.21	4.04	2.69	2.99
MEAN				7.71	56.9	1.57	3.90	17.6	19.9	1.61	0.84	3.27	5.22	4.30	2.68	2.96
100	200	Lipton	with		m.v.	1.25	4.33	16.3	21.8	1.88	0.82	3.41	5.17	4.87	2.72	2.40
100	200	Lipton	with		54.6	0.89	3.85	17.0	19.6	1.57	0.95	3.41	5.15	4.26	2.66	2.83
100	200	Lipton	with		49.8	1.65	3.38	21.0	19.2	1.48	1.91	3.31	5.25	4.07	2.68	1.95
100	200	Lipton	with		50.7	0.92	4.36	20.5	22.9	1.87	1.36		5.24	4.35	2.70	2.31
MEAN				7.60	51.7	1.18	3.98	18.7	20.9	1.70	1.28	3.36	5.20	4.39	2.68	2.37

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds				Yield t ha ⁻¹	
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P		Ca
2002/2003															
0	100	<i>Bristol</i>	with	5.51	36.6	3.93	3.07	14.2	18.0	1.20	1.45	3.14	5.43	4.37	2.5701
0	100	<i>Bristol</i>	with	5.60	35.0	3.72	3.26	15.6	16.9	1.18	2.21	3.00	5.69	4.38	2.5844
0	100	<i>Bristol</i>	with	4.83	39.5	3.61	2.83	12.2	15.5	1.09	0.75	3.32	5.63	4.31	2.5636
0	100	<i>Bristol</i>	with	—	28.9	3.19	2.69	12.7	15.3	0.99	1.16	2.97	5.97	4.21	2.6867
MEAN				—	35.0	3.61	2.96	13.7	16.4	1.12	1.39	3.11	5.68	4.32	2.59
0	100	<i>Bristol</i>	without	6.07	39.9	4.16	3.08	17.3	17.1	1.07	2.49	3.03	5.77	4.28	2.5554
0	100	<i>Bristol</i>	without	5.00	39.9	4.61	2.55	12.8	14.5	0.97	1.19	2.76	6.19	3.85	2.4803
0	100	<i>Bristol</i>	without	4.91	40.8	4.06	2.63	13.0	15.1	1.06	0.77	3.03	5.90	4.01	2.6116
0	100	<i>Bristol</i>	without	—	39.0	3.72	2.52	12.9	12.6	0.88	0.86	3.14	5.34	4.27	2.5137
MEAN				—	39.9	4.14	2.70	14.0	14.8	1.00	1.33	2.99	5.80	4.10	2.54
0	100	<i>Lipton</i>	with	4.61	33.9	3.19	3.52	13.3	15.7	1.01	1.15	3.19	5.40	4.32	2.7146
0	100	<i>Lipton</i>	with	4.00	37.1	2.10	3.11	12.5	14.7	0.92	0.91	2.79	5.87	4.24	2.6448
0	100	<i>Lipton</i>	with	3.58	33.8	2.50	2.87	11.6	12.8	0.89	1.82	3.12	5.56	4.04	2.5490
0	100	<i>Lipton</i>	with	—	34.9	2.19	2.82	14.7	12.7	0.94	3.10	2.77	5.97	4.12	2.5473
MEAN				—	34.9	2.50	3.08	13.0	14.0	0.94	1.75	2.97	5.70	4.18	2.61
0	100	<i>Lipton</i>	without	4.68	36.2	2.99	3.08	13.5	12.6	0.98	0.96	3.10	5.78	4.12	2.6123
0	100	<i>Lipton</i>	without	4.58	31.1	2.66	2.93	15.1	13.2	0.94	2.78	2.60	5.93	3.95	2.4960
0	100	<i>Lipton</i>	without	3.62	36.3	2.70	2.93	10.3	11.8	0.93	0.47	2.97	5.49	3.90	2.5783
0	100	<i>Lipton</i>	without	—	41.1	3.14	3.44	15.4	14.4	1.03	2.92	3.37	5.33	4.30	2.7113
MEAN				—	36.2	2.87	3.10	13.5	13.0	0.97	1.78	3.01	5.63	4.07	2.60
0	200	<i>Bristol</i>	with	4.78	44.4	1.00	3.28	13.7	18.2	1.11	2.31	2.84	4.87	4.17	2.4871
0	200	<i>Bristol</i>	with	4.74	44.1	1.61	2.84	13.0	15.8	1.07	2.12	3.09	5.08	4.19	2.4433
0	200	<i>Bristol</i>	with	5.17	38.7	2.21	3.53	15.2	17.9	1.35	2.97	3.24	5.08	3.69	2.6529
0	200	<i>Bristol</i>	with	—	50.3	2.20	2.96	14.9	15.9	1.18	3.88	3.02	6.16	4.23	2.8032
MEAN				—	44.4	1.75	3.15	14.2	16.9	1.18	2.82	3.05	5.30	4.07	2.59
0	200	<i>Bristol</i>	without	4.19	45.9	0.77	3.12	14.5	14.0	1.01	2.42	2.97	5.37	4.18	2.5436
0	200	<i>Bristol</i>	without	4.84	45.2	2.60	2.88	13.0	15.8	1.14	3.07	2.56	5.79	3.70	2.5170
0	200	<i>Bristol</i>	without	4.78	41.3	2.39	3.01	14.2	16.8	1.32	3.18	3.13	5.49	3.66	2.6543
0	200	<i>Bristol</i>	without	—	43.7	1.40	3.13	13.0	14.6	1.03	1.57	2.95	4.92	3.81	2.4035
MEAN				—	44.0	1.79	3.04	13.7	15.3	1.13	2.56	2.90	5.39	3.84	2.53

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds						Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg	
2002/2003																
0	200	<i>Lipton</i>	with	3.78	42.6	1.13	3.46	12.4	14.7	1.03	1.45	2.97	5.31	4.07	2.69	4.00
0	200	<i>Lipton</i>	with	3.23	44.4	1.04	3.36	12.0	11.5	0.96	0.95	3.06	5.84	3.96	2.64	3.83
0	200	<i>Lipton</i>	with	3.65	43.2	1.52	3.28	11.7	12.3	0.95	0.72	3.36	5.37	4.12	2.65	3.72
0	200	<i>Lipto2.91</i>	with	—	43.7	1.13	2.89	10.5	11.0	0.93	1.04	3.28	5.14	3.87	2.6994	—
MEAN				4.00	43.5	1.20	3.25	11.7	12.4	0.97	1.04	3.17	5.41	4.01	2.67	3.62
0	200	<i>Lipton</i>	without	4.00	41.1	1.69	3.31	14.5	12.6	0.98	2.69	3.12	5.42	3.77	2.68	3.50
0	200	<i>Lipton</i>	without	3.50	43.9	1.36	3.06	15.2	12.5	0.95	2.59	2.83	5.61	3.41	2.50	2.00
0	200	<i>Lipton</i>	without	3.71	43.3	1.64	3.05	12.2	12.9	1.05	1.33	3.35	5.33	3.89	2.68	3.78
0	200	<i>Lipton</i>	without	—	43.1	1.21	3.55	13.9	15.1	1.13	1.66	3.23	5.32	3.76	2.9449	—
MEAN				6.46	42.9	1.48	3.24	14.0	13.3	1.03	2.07	3.13	5.42	3.71	2.60	3.19
100	100	<i>Bristol</i>	with	6.46	40.0	4.81	2.92	14.0	16.7	1.07	0.32	3.38	5.72	4.51	2.47	2.85
100	100	<i>Bristol</i>	with	6.72	45.0	5.28	3.25	14.1	16.9	1.34	0.27	3.37	5.64	4.49	2.47	3.58
100	100	<i>Bristol</i>	with	5.58	37.2	5.38	2.59	11.9	15.3	0.97	0.25	3.38	5.35	4.32	2.58	2.47
100	100	<i>Bristol87</i>	with	—	40.8	4.55	2.79	15.2	19.4	1.16	0.30	3.12	5.98	4.43	2.5741	—
MEAN				6.85	40.8	5.00	2.89	13.8	17.1	1.14	0.28	3.31	5.67	4.44	2.52	2.58
100	100	<i>Bristol</i>	without	6.09	39.3	5.99	2.94	14.5	14.8	1.14	0.11	3.04	6.08	4.14	2.56	1.72
100	100	<i>Bristol</i>	without	6.71	35.5	4.90	2.88	14.1	16.6	1.24	0.34	3.34	5.53	4.02	2.64	1.59
100	100	<i>Bristol</i>	without	—	36.4	4.33	2.89	12.1	13.4	1.08	0.03	3.44	5.32	4.61	2.6172	1.96
MEAN				5.32	37.1	4.82	2.98	14.2	15.3	1.12	0.19	3.24	5.66	4.20	2.61	2.00
100	100	<i>Lipton</i>	with	5.30	37.0	3.35	3.41	13.9	18.1	1.04	0.56	3.37	5.57	4.33	2.56	3.49
100	100	<i>Lipton</i>	with	4.41	46.3	3.57	3.33	12.5	17.2	1.11	0.26	3.30	5.54	4.26	2.56	3.21
100	100	<i>Lipton</i>	with	—	54.4	2.63	2.72	11.6	12.8	0.92	0.23	3.33	5.62	4.21	2.68	3.00
100	100	<i>Lipto4.44</i>	with	—	43.4	3.17	3.09	12.1	15.5	1.03	0.30	3.00	6.10	4.08	2.7055	—
MEAN				5.18	33.9	5.87	2.93	14.7	14.5	0.91	0.55	3.25	5.71	4.22	2.62	2.81
100	100	<i>Lipton</i>	without	5.21	31.9	3.62	3.16	12.8	13.7	1.16	0.22	2.93	5.74	4.10	2.60	2.30
100	100	<i>Lipton</i>	without	4.22	35.5	3.45	2.93	11.4	13.9	1.01	0.06	2.85	5.91	4.07	2.60	2.69
100	100	<i>Lipton</i>	without	—	37.8	2.60	3.20	12.3	13.1	0.91	0.17	3.47	5.19	4.63	2.6288	—
MEAN				34.8	34.8	3.88	3.06	12.8	13.8	1.00	0.25	3.10	5.65	4.28	2.65	2.54

Table A.11: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves						Seeds						Yield t ha ⁻¹
				S	N	SO ₄ -S	P	K	Ca	Mg	Cl	S	P	Ca	Mg	
2002/2003																
100	200	<i>Bristol</i>	with	6.37	48.5	3.77	2.71	10.1	16.2	1.10	0.17	3.55	5.31	4.33	2.49	4.07
100	200	<i>Bristol</i>	with	5.87	47.4	4.95	3.06	11.8	15.4	1.12	0.06	3.49	5.16	4.27	2.48	2.99
100	200	<i>Bristol</i>	with	5.26	41.5	4.47	3.00	11.6	16.0	1.28	0.06	3.54	5.06	3.94	2.64	2.70
100	200	<i>Bristol</i>	with	—	43.7	3.20	2.87	10.0	12.6	1.07	0.02	3.64	5.03	4.30	2.688	—
MEAN				—	45.3	4.10	2.91	10.9	15.0	1.14	0.08	3.55	5.14	4.21	2.57	3.16
100	200	<i>Bristol</i>	without	5.90	44.3	3.86	2.72	11.9	14.9	1.03	0.30	3.39	5.38	4.18	2.67	2.95
100	200	<i>Bristol</i>	without	5.63	46.6	3.38	3.16	11.6	16.0	1.11	0.06	3.38	5.56	3.98	2.53	3.27
100	200	<i>Bristol</i>	without	5.40	43.5	5.36	2.88	10.9	14.8	1.29	0.00	3.71	5.58	3.45	2.64	2.91
100	200	<i>Bristol</i>	without	—	42.9	3.93	3.17	13.9	14.9	1.15	0.12	3.75	5.09	4.04	2.920	—
MEAN				—	44.3	4.13	2.98	12.1	15.1	1.15	0.12	3.55	5.40	3.91	2.58	3.08
100	200	<i>Lipton</i>	with	4.71	48.6	2.53	3.15	11.0	13.4	0.86	0.18	3.55	5.25	4.23	2.66	4.32
100	200	<i>Lipton</i>	with	4.77	44.1	2.44	3.48	13.3	14.0	0.99	0.17	3.19	5.36	3.93	2.55	3.87
100	200	<i>Lipton</i>	with	3.98	33.9	2.23	3.12	10.4	13.4	0.99	0.12	3.65	5.15	3.96	2.65	3.78
100	200	<i>Lipton</i>	with	—	46.3	1.87	3.24	11.2	16.3	1.32	0.16	3.70	5.94	3.77	2.7069	—
MEAN				—	43.2	2.27	3.25	11.5	14.3	1.04	0.16	3.52	5.43	3.97	2.64	3.42
100	200	<i>Lipton</i>	without	5.44	42.4	3.48	3.43	13.4	16.4	1.12	0.24	3.36	5.48	4.05	2.68	3.20
100	200	<i>Lipton</i>	without	4.36	40.0	2.84	2.92	12.2	12.5	0.89	0.16	3.18	5.40	3.52	2.48	2.33
100	200	<i>Lipton</i>	without	4.53	39.2	3.40	2.87	10.1	14.1	1.03	0.17	3.41	5.75	3.69	2.68	3.38
100	200	<i>Lipton</i>	without	—	44.8	2.63	3.40	11.3	12.9	1.01	0.12	3.72	5.05	4.16	2.667	—
MEAN				—	41.6	3.09	3.16	11.7	14.0	1.01	0.17	3.42	5.42	3.85	2.63	3.15

note: n.a. – not available; m.v. – missing value

Table A.12: Mineral nutrient content in straw and pod wall samples, Braunschweig, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls						
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg
2000/2001														
0	100	<i>Bristol</i>	with	4.58	2.73	25.3	15.2	1.04	2.76	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bris402</i>	with		2.96	24.4	14.0	1.14	6.88	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bris406</i>	with		2.88	22.5	13.3	0.99	5.17	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bris405</i>	with		2.81	27.5	10.9	0.91	4.69	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			4.70	2.85	24.9	13.3	1.02	4.87	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bristol</i>	4x6thout		2.81	22.2	13.3	1.03	2.55	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bristol</i>	4x7thout		2.83	22.9	16.6	1.14	6.44	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bristol</i>	3x9thout		2.42	24.2	11.3	0.95	5.42	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Bristol</i>	4x6thout		2.74	23.1	12.3	0.90	2.91	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			4.50	2.70	23.1	13.4	1.01	4.33	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lip0071</i>	with		3.58	24.7	8.7	1.09	3.52	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lip0009</i>	with		2.93	25.8	11.8	0.99	3.60	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lip0099</i>	with		2.92	25.0	11.5	0.98	3.11	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lip0037</i>	with		2.96	26.9	12.0	0.98	2.78	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			5.04	3.10	25.6	11.0	1.01	3.25	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lipton</i>	5x6thout		3.65	28.7	12.5	1.27	4.86	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lipton</i>	4x9thout		2.75	28.2	11.4	0.94	2.80	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lipton</i>	4x8thout		2.48	24.9	13.3	0.89	3.54	n.a.	n.a.	n.a.	n.a.	n.a.
0	100	<i>Lipton</i>	4x9thout		2.88	27.0	11.4	0.93	2.03	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			5.11	2.94	27.2	12.1	1.01	3.31	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bris405</i>	with		2.39	26.2	16.4	1.23	7.00	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bris408</i>	with		2.71	22.6	12.9	1.02	3.40	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bris400</i>	with		2.82	26.7	10.4	0.88	2.02	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bris400</i>	with		2.78	26.9	12.0	1.02	5.13	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			4.68	2.68	25.6	12.9	1.04	4.39	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bristol</i>	5x20thout		3.13	25.8	14.9	1.23	6.42	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bristol</i>	4x4thout		2.57	23.9	11.1	0.97	5.09	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bristol</i>	4x6thout		2.82	25.4	11.0	0.91	2.16	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	<i>Bristol</i>	5x0thout		3.15	25.9	10.7	0.95	2.76	n.a.	n.a.	n.a.	n.a.	n.a.
	MEAN			4.85	2.92	25.2	11.9	1.02	4.10	n.a.	n.a.	n.a.	n.a.	n.a.

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls						
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg
2000/2001														
0	200	Lipton	with	3.94	2.44	22.3	11.2	0.85	3.41	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	without		2.91	24.6	10.7	0.95	3.59	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	with		2.92	25.2	8.61	0.93	1.84	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	without		2.61	27.0	12.7	0.93	2.42	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				4.51	2.72	24.8	10.8	0.92	2.81	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	with		3.61	25.5	12.6	1.16	5.30	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	without		3.58	27.1	11.3	1.21	4.85	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	with		2.45	27.6	12.7	0.86	2.43	n.a.	n.a.	n.a.	n.a.	n.a.
0	200	Lipton	without		3.02	26.9	10.6	0.91	2.83	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				5.17	3.17	26.8	11.8	1.04	3.85	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	with		3.12	27.6	13.6	1.11	2.35	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	without		3.24	27.3	13.4	1.06	2.87	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	with		2.81	25.2	11.4	0.99	4.64	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	without		2.67	28.3	12.4	0.97	6.04	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				5.00	2.96	27.1	12.7	1.03	3.98	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	with		2.67	21.8	13.8	1.02	2.39	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	without		2.85	25.5	11.8	1.13	5.14	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	with		2.46	25.7	14.0	1.06	3.90	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Bristol	without		3.26	28.3	10.4	1.01	3.02	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				4.80	2.81	25.3	12.5	1.06	3.61	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	with		3.45	27.0	14.6	1.18	6.80	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	without		3.17	23.8	11.6	1.04	3.24	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	with		2.6	27.0	12.6	0.97	3.24	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	without		2.7	26.1	12.8	0.95	6.09	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				5.10	2.98	26.0	12.9	1.04	4.84	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	with		3.51	24.9	10.9	1.26	4.37	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	without		3.55	25.8	11.2	1.21	3.31	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	with		3.08	24.6	10.3	0.92	2.69	n.a.	n.a.	n.a.	n.a.	n.a.
150	100	Lipton	without		2.79	26.7	11.4	0.87	5.65	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				5.21	3.23	25.5	11.0	1.07	4.01	n.a.	n.a.	n.a.	n.a.	n.a.

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls						
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg
2000/2001														
150	200	<i>Bristol</i>	with	4.84	2.62	24.0	12.5	0.99	3.30	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	without		2.99	24.2	11.4	0.99	4.84	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	with		2.79	29.4	12.4	1.01	6.22	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	with		2.74	26.1	12.5	0.83	2.17	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				4.74	2.79	25.9	12.2	0.96	4.13	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	with		2.77	28.6	15.7	1.26	3.91	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	without		3.18	25.9	11.0	1.03	5.97	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	with		2.56	27.2	12.2	0.92	4.51	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Bristol</i>	with		2.86	26.2	13.5	1.01	7.08	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				4.79	2.84	27.0	13.1	1.06	5.37	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	with		2.76	20.6	11.5	0.98	2.51	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	with		3.33	25.3	9.8	1.13	2.12	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	with		2.57	28.1	11.5	0.99	2.71	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	with		2.69	24.4	11.4	0.90	5.90	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				4.58	2.84	24.6	11.0	1.00	3.31	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	without		3.85	31.8	12.4	1.20	2.75	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	without		3.26	28.4	10.1	1.04	2.63	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	with		2.88	24.3	12.3	0.99	3.00	n.a.	n.a.	n.a.	n.a.	n.a.
150	200	<i>Lipton</i>	without		3.32	28.1	12.0	1.07	4.97	n.a.	n.a.	n.a.	n.a.	n.a.
MEAN				5.36	3.33	28.1	11.7	1.08	3.34	n.a.	n.a.	n.a.	n.a.	n.a.
2001/2002														
0	100	<i>Bristol</i>	with	1.55	1.87	10.4	11.1	0.89	5.236	1.70	2.28	14.7	0.69	0.15
0	100	<i>Bristol</i>	with	1.47	1.79	3.65	14.9	0.69	0.661	1.97	2.01	21.2	0.61	0.06
0	100	<i>Bristol</i>	with	1.68	1.92	6.72	14.0	0.84	3.082	1.39	1.64	18.5	0.54	0.07
0	100	<i>Bristol</i>	with	1.42	1.72	7.41	13.8	0.72	3.003	1.30	1.57	18.2	0.48	0.03
MEAN				1.53	1.83	7.04	13.4	0.79	2.98	1.21	1.88	18.2	0.58	0.08

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw					Pod walls					
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg
2001/2002														
0	100	<i>Bristol</i>	without	1.75	1.97	9.16	13.7	0.85	41.422	1.42	1.88	16.5	0.57	0.10
0	100	<i>Bristol</i>	without	1.34	1.79	2.34	14.6	0.63	01323	1.28	1.95	16.7	0.55	0.16
0	100	<i>Bristol</i>	without	1.80	2.07	7.45	13.3	0.81	21620	1.69	1.99	17.7	0.61	0.08
0	100	<i>Bristol</i>	without	1.08	1.35	2.72	12.1	0.52	01724	1.22	2.14	16.1	0.57	0.08
	MEAN			1.49	1.80	5.42	13.4	0.70	2.02	1.40	1.99	16.8	0.58	0.11
0	100	<i>Lipton</i>	with	1.85	1.48	5.96	12.1	0.71	21497	1.07	1.70	16.6	0.52	0.10
0	100	<i>Lipton</i>	with	2.16	2.43	8.75	17.6	0.91	31286	1.29	1.89	19.3	0.52	0.13
0	100	<i>Lipton</i>	with	1.45	1.66	4.59	14.8	0.72	11695	1.19	1.75	17.9	0.57	0.07
0	100	<i>Lipton</i>	with	1.99	2.27	4.27	15.5	0.69	01980	1.28	1.95	18.9	0.52	0.08
	MEAN			1.86	1.96	5.89	15.0	0.76	2.11	1.21	1.82	18.2	0.53	0.09
0	100	<i>Lipton</i>	without	1.77	1.84	4.74	14.8	0.73	10904	0.91	1.60	16.0	0.52	0.08
0	100	<i>Lipton</i>	without	1.59	1.84	4.51	13.9	0.64	11600	1.06	1.52	18.0	0.51	0.09
0	100	<i>Lipton</i>	without	1.73	1.73	4.29	15.0	0.62	11407	1.17	1.71	19.3	0.51	0.11
0	100	<i>Lipton</i>	without	1.48	1.43	5.22	12.5	0.61	11926	1.39	1.94	18.4	0.62	0.08
	MEAN			1.64	1.71	4.69	14.0	0.65	1.74	1.13	1.69	17.9	0.54	0.09
0	200	<i>Bristol</i>	with	1.74	2.00	6.31	13.9	0.84	21043	1.11	1.81	15.0	0.57	0.12
0	200	<i>Bristol</i>	with	1.71	1.87	6.75	11.9	0.75	31015	1.27	1.90	15.3	0.53	0.10
0	200	<i>Bristol</i>	with	1.41	1.67	2.73	11.4	0.65	01320	1.42	2.05	15.6	0.60	0.10
0	200	<i>Bristol</i>	with	1.71	1.60	11.1	12.1	0.76	41222	1.40	1.89	16.6	0.52	0.11
	MEAN			1.64	1.79	6.73	12.4	0.75	2.41	1.30	1.91	15.6	0.56	0.11
0	200	<i>Bristol</i>	without	1.44	1.47	5.06	11.4	0.72	11567	1.43	1.91	17.8	0.58	0.12
0	200	<i>Bristol</i>	without	1.74	2.09	4.34	12.8	0.72	01847	1.61	2.22	15.6	0.64	0.08
0	200	<i>Bristol</i>	without	1.64	2.07	3.52	13.0	0.67	01523	1.26	1.86	17.4	0.57	0.05
0	200	<i>Bristol</i>	without	1.96	2.39	4.83	14.2	0.78	11080	1.47	2.03	16.7	0.56	0.01
	MEAN			1.70	2.01	4.44	12.8	0.72	0.99	1.44	2.01	16.8	0.59	0.07
0	200	<i>Lipton</i>	with	2.93	1.90	16.4	13.0	1.13	81365	1.04	1.90	15.5	0.54	0.12
0	200	<i>Lipton</i>	with	1.55	1.83	4.87	13.8	0.75	10399	0.95	1.73	16.4	0.49	0.09
0	200	<i>Lipton</i>	with	1.51	1.90	15.2	8.4	0.92	71497	0.98	1.74	14.1	0.58	0.12
0	200	<i>Lipton</i>	with	1.71	1.56	8.51	13.8	0.64	31244	1.30	1.96	17.4	0.47	0.20
	MEAN			1.93	1.80	11.2	12.2	0.86	5.10	1.07	1.83	15.9	0.52	0.13

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw						Pod walls					
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg	Cl
2001/2002															
0	200	<i>Lipton</i>	without	1.73	1.92	5.81	13.8	0.74	10499	0.86	1.57	14.8	0.50	0.07	
0	200	<i>Lipton</i>	without	2.06	1.96	10.5	13.2	0.95	41069	1.09	1.62	16.8	0.51	0.10	
0	200	<i>Lipton</i>	without	1.64	2.05	5.70	13.7	0.67	11305	1.01	1.77	14.8	0.48	0.07	
0	200	<i>Lipton</i>	without	1.57	1.95	10.9	9.5	0.84	31794	1.21	1.67	16.0	0.54	0.04	
MEAN				1.75	1.97	8.24	12.6	0.80	2.68	1.04	1.66	15.6	0.51	0.07	
150	100	<i>Bristol</i>	with	1.54	1.96	2.56	14.1	0.61	01140	1.72	1.95	18.2	0.57	0.07	
150	100	<i>Bristol</i>	with	1.85	2.48	4.42	16.5	0.80	01344	1.17	1.69	17.8	0.53	0.04	
150	100	<i>Bristol</i>	with	2.66	2.41	16.1	13.9	1.01	11563	1.60	1.94	19.9	0.64	0.03	
150	100	<i>Bristol</i>	with	2.40	2.12	12.6	11.6	0.92	21232	1.83	2.15	19.5	0.63	0.02	
MEAN				2.11	2.24	8.91	14.0	0.84	1.06	1.58	1.93	18.8	0.59	0.04	
150	100	<i>Bristol</i>	without	1.68	2.11	2.71	14.6	0.73	01138	1.80	2.21	16.5	0.65	0.07	
150	100	<i>Bristol</i>	without	1.59	2.19	2.46	15.5	0.70	01145	2.03	2.14	18.9	0.67	0.06	
150	100	<i>Bristol</i>	without	2.38	2.11	11.8	15.3	1.01	21080	1.73	2.01	20.3	0.62	0.04	
150	100	<i>Bristol</i>	without	1.37	1.58	2.36	12.1	0.53	01143	2.14	1.89	22.1	0.55	0.05	
MEAN				1.76	2.00	4.84	14.4	0.74	0.61	1.93	2.06	19.5	0.62	0.06	
150	100	<i>Lipton</i>	with	2.54	2.08	7.92	15.8	0.81	11404	1.23	1.61	17.0	0.49	0.09	
150	100	<i>Lipton</i>	with	2.02	1.57	5.86	13.2	0.70	01646	1.27	1.95	15.6	0.54	0.06	
150	100	<i>Lipton</i>	with	1.58	1.63	4.59	12.4	0.68	01630	1.35	1.84	19.7	0.61	0.06	
150	100	<i>Lipton</i>	with	3.36	2.58	14.5	14.2	0.74	31582	1.68	2.24	18.6	0.59	0.03	
MEAN				2.38	1.97	8.22	13.9	0.73	1.56	1.38	1.91	17.7	0.56	0.06	
150	100	<i>Lipton</i>	without	1.75	1.71	3.57	15.2	0.63	01342	1.11	1.64	16.5	0.52	0.03	
150	100	<i>Lipton</i>	without	2.97	2.18	13.3	14.6	0.94	21704	0.91	1.55	16.7	0.54	0.03	
150	100	<i>Lipton</i>	without	1.67	1.63	2.88	15.0	0.61	01242	1.22	1.85	17.3	0.51	0.09	
150	100	<i>Lipton</i>	without	3.02	2.43	14.1	15.0	0.86	21849	1.32	1.80	20.0	0.50	0.04	
MEAN				2.35	1.99	8.47	14.9	0.76	1.53	1.14	1.71	17.6	0.52	0.05	
150	200	<i>Bristol</i>	with	1.79	2.24	2.79	13.3	0.71	01047	1.77	2.22	13.9	0.55	0.20	
150	200	<i>Bristol</i>	with	2.17	2.09	4.47	14.3	0.71	01434	1.86	2.12	18.4	0.59	0.05	
150	200	<i>Bristol</i>	with	2.28	2.39	10.3	13.4	0.96	11134	1.23	2.00	15.1	0.52	0.04	
150	200	<i>Bristol</i>	with	1.82	1.71	8.86	11.7	0.67	11342	1.67	1.93	18.7	0.54	0.03	
MEAN				2.02	2.11	6.60	13.2	0.76	0.73	1.42	2.07	16.5	0.55	0.08	

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls							
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg	Cl
				mg g ⁻¹											
				2001/2002											
150	200	<i>Bristol</i>	without	2.10	2.04	5.42	14.4	0.73	0.17	0.64	1.57	2.27	14.8	0.63	0.06
150	200	<i>Bristol</i>	without	2.18	2.54	4.69	14.5	0.81	0.14	0.94	2.31	2.35	20.6	0.67	0.06
150	200	<i>Bristol</i>	without	1.95	2.33	5.10	13.3	0.76	0.25	0.54	1.66	2.05	16.5	0.61	0.04
150	200	<i>Bristol</i>	without	2.33	2.28	6.99	13.9	0.75	0.56	0.66	1.21	1.96	17.5	0.51	0.03
MEAN				2.14	2.30	5.55	14.0	0.76	0.50	0.50	1.69	2.16	17.4	0.61	0.05
150	200	<i>Lipton</i>	with	2.28	2.45	4.09	17.8	0.74	0.25	0.25	1.01	1.75	17.3	0.54	0.05
150	200	<i>Lipton</i>	with	1.93	1.85	6.01	13.2	0.68	0.74	0.48	1.04	1.92	17.2	0.54	0.13
150	200	<i>Lipton</i>	with	1.99	1.72	5.19	12.7	0.66	0.74	0.58	1.16	2.06	17.9	0.59	0.09
150	200	<i>Lipton</i>	with	3.19	2.29	19.0	10.8	0.90	1.68	0.66	1.67	2.51	19.3	0.62	0.05
MEAN				2.35	2.08	8.58	13.6	0.75	0.86	0.86	1.22	2.06	17.9	0.57	0.08
150	200	<i>Lipton</i>	without	2.83	2.29	9.47	16.6	0.87	1.96	0.66	1.22	2.16	17.3	0.56	0.11
150	200	<i>Lipton</i>	without	2.40	2.47	5.43	15.9	0.81	0.63	0.43	1.24	2.04	18.2	0.63	0.06
150	200	<i>Lipton</i>	without	2.13	2.11	8.66	13.3	0.78	1.43	0.35	1.31	1.82	18.0	0.53	0.05
150	200	<i>Lipton</i>	without	2.26	1.90	6.51	14.7	0.67	0.65	0.15	1.21	1.87	20.3	0.49	0.04
MEAN				2.41	2.19	7.52	15.1	0.78	1.02	1.02	1.25	1.97	18.5	0.55	0.06
				2002/2003											
0	100	<i>Bristol</i>	with	1.92	1.12	30.1	10.9	1.15	15.4	15.4	1.15	27.5	19.9	1.77	9.43
0	100	<i>Bristol</i>	with	—	1.20	29.0	13.2	1.27	16.0	16.0	1.70	24.0	18.9	1.87	10.7
0	100	<i>Bristol</i>	with	—	1.18	29.5	15.0	1.69	10.9	10.9	1.19	29.0	21.6	2.54	8.20
0	100	<i>Bristol</i>	with	—	0.90	27.3	15.6	1.38	11.2	11.2	1.35	22.5	19.6	2.06	9.74
MEAN				—	1.10	29.0	13.7	1.37	13.4	13.4	1.35	25.7	20.0	2.06	9.51
0	100	<i>Bristol</i>	without	—	1.21	31.9	14.0	1.23	14.0	14.0	1.15	25.5	19.5	1.57	9.11
0	100	<i>Bristol</i>	without	—	1.21	31.3	14.7	1.29	14.2	14.2	1.33	20.7	20.7	1.77	9.95
0	100	<i>Bristol</i>	without	—	1.40	31.5	10.9	1.39	16.7	16.7	1.07	23.8	17.4	2.10	6.63
0	100	<i>Bristol</i>	without	—	1.12	27.8	11.4	1.18	11.9	11.9	1.24	21.2	19.6	1.75	5.40
MEAN				—	1.24	30.6	12.8	1.27	14.2	14.2	1.20	22.8	19.3	1.80	7.77

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls							
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg	Cl
2002/2003															
0	100	Lipton	with	3.07	0.95	31.8	15.8	1.39	11.0	6.22	1.07	27.6	20.0	2.24	8.28
0	100	Lipton	59 with		0.98	28.3	11.5	1.27	13.6	4.01	0.94	23.3	19.6	2.04	10.5
0	100	Lipton	10 with		1.10	27.6	12.7	1.17	13.3	3.67	1.22	23.0	22.2	2.19	9.23
0	100	Lipton	54 with		<u>1.41</u>	30.4	<u>12.6</u>	<u>1.45</u>	<u>15.1</u>	5.08	<u>1.21</u>	<u>23.5</u>	<u>19.9</u>	<u>2.30</u>	<u>9.43</u>
MEAN					1.11	29.5	13.2	1.32	13.3	4.75	1.11	24.3	20.4	2.19	9.36
0	100	Lipton	with		1.01	29.1	12.5	1.35	15.5	4.18	0.90	25.9	19.3	1.97	10.1
0	100	Lipton	with		0.85	27.3	11.8	1.11	13.3	4.38	1.13	26.7	21.4	1.97	9.14
0	100	Lipton	with		0.88	28.3	11.8	1.35	11.9	4.94	1.34	27.8	22.7	2.52	10.0
0	100	Lipton	with		<u>1.11</u>	<u>30.2</u>	<u>15.1</u>	<u>1.51</u>	<u>12.2</u>	<u>5.17</u>	<u>1.27</u>	<u>30.0</u>	<u>23.1</u>	<u>2.88</u>	<u>7.49</u>
MEAN					0.96	28.7	12.8	1.33	13.2	4.67	1.16	27.6	21.6	2.34	9.19
0	200	Bristol	19 with		0.94	30.1	14.4	1.20	13.0	4.09	0.81	25.4	17.6	1.73	9.68
0	200	Bristol	97 with		0.91	29.5	16.5	1.59	10.0	3.53	1.25	26.6	23.6	2.27	8.53
0	200	Bristol	68 with		0.92	28.1	11.2	1.22	13.2	3.68	0.82	22.9	17.0	1.73	7.23
0	200	Bristol	20 with		<u>1.14</u>	<u>31.2</u>	<u>12.3</u>	<u>1.55</u>	<u>13.5</u>	<u>3.53</u>	<u>1.28</u>	<u>27.0</u>	<u>19.7</u>	<u>2.32</u>	<u>7.09</u>
MEAN					0.98	29.7	13.6	1.39	12.4	3.71	1.04	25.5	19.5	2.01	8.14
0	200	Bristol	with		1.31	28.3	15.9	1.37	10.2	2.41	1.48	26.2	23.9	1.99	8.41
0	200	Bristol	with		1.08	29.1	14.4	1.51	10.6	2.82	1.18	21.9	21.3	2.38	6.86
0	200	Bristol	with		1.13	28.8	12.8	1.32	10.4	3.17	1.09	23.2	20.6	1.62	6.24
0	200	Bristol	with		<u>1.06</u>	<u>32.7</u>	<u>10.1</u>	<u>1.11</u>	<u>13.3</u>	<u>4.52</u>	<u>1.03</u>	<u>24.5</u>	<u>18.5</u>	<u>2.39</u>	<u>7.41</u>
MEAN					1.15	29.7	13.3	1.33	11.1	3.23	1.20	23.9	21.1	2.10	7.23
0	200	Lipton	81 with		0.85	31.1	10.9	1.21	12.8	4.40	0.74	27.6	20.2	1.89	8.34
0	200	Lipton	37 with		0.81	25.7	12.1	1.37	9.34	6.45	1.02	30.5	23.7	2.79	9.90
0	200	Lipton	59 with		1.09	28.1	8.74	1.00	8.97	8.17	0.97	28.7	18.5	2.26	4.76
0	200	Lipton	36 with		<u>1.19</u>	<u>30.9</u>	<u>17.4</u>	<u>1.73</u>	<u>12.5</u>	<u>4.51</u>	<u>0.94</u>	<u>26.7</u>	<u>20.3</u>	<u>2.10</u>	<u>8.76</u>
MEAN					0.99	29.0	12.3	1.33	10.9	5.88	0.92	28.4	20.7	2.26	7.94
0	200	Lipton	with		0.76	27.7	11.8	1.26	9.62	3.80	0.79	27.0	19.1	2.11	7.14
0	200	Lipton	with		1.17	31.0	16.6	1.61	13.1	3.66	1.28	27.4	21.7	2.00	7.10
0	200	Lipton	with		1.08	31.1	9.79	1.39	12.6	3.04	0.93	30.3	17.9	2.59	10.6
0	200	Lipton	with		<u>1.08</u>	<u>30.5</u>	<u>10.9</u>	<u>1.14</u>	<u>12.4</u>	<u>3.42</u>	<u>0.88</u>	<u>20.7</u>	<u>14.6</u>	<u>1.77</u>	<u>6.48</u>
MEAN					1.02	30.1	12.3	1.35	11.9	3.48	0.97	26.3	18.3	2.12	7.82

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls							
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg	Cl
2002/2003															
150	100	<i>Bristol</i>	with	4.77	0.88	31.4	10.5	1.15	6.51	11.1	0.97	27.4	19.0	2.41	1.48
150	100	<i>BrisolB5</i>	with		0.93	32.3	13.9	1.55	2.11	10.5	1.27	27.5	23.2	2.81	1.87
150	100	<i>BrisolA1</i>	with		1.15	33.4	12.3	1.42	2.97	11.8	1.30	36.1	21.5	2.48	2.13
150	100	<i>BrisolB6</i>	with		<u>1.12</u>	<u>36.7</u>	<u>8.96</u>	<u>1.15</u>	<u>4.43</u>	<u>8.69</u>	<u>1.07</u>	<u>30.7</u>	<u>20.1</u>	<u>2.49</u>	<u>1.53</u>
MEAN					1.02	33.4	11.4	1.32	4.01	10.5	1.15	30.4	21.0	2.55	1.75
150	100	<i>Bristol</i>	with		1.01	34.2	9.30	1.14	2.80	10.9	1.02	27.9	15.4	1.91	1.24
150	100	<i>Bristol</i>	with		1.15	32.7	11.0	1.11	2.83	9.25	1.28	25.6	19.0	2.02	1.64
150	100	<i>Bristol</i>	with		1.06	30.0	13.1	1.27	12.1	6.37	1.28	28.1	25.7	2.31	6.63
150	100	<i>Bristol</i>	with		<u>1.13</u>	<u>34.8</u>	<u>8.62</u>	<u>1.14</u>	<u>3.83</u>	<u>9.89</u>	<u>1.08</u>	<u>40.2</u>	<u>18.9</u>	<u>2.40</u>	<u>2.90</u>
MEAN					1.09	32.9	10.5	1.17	5.39	9.11	1.17	30.4	19.8	2.16	3.10
150	100	<i>Lipton00</i>	with		0.91	32.3	11.3	1.09	3.53	12.8	1.07	30.2	23.4	2.04	2.56
150	100	<i>Lipton91</i>	with		1.19	33.1	18.5	2.05	3.40	12.0	1.01	32.8	20.8	2.50	2.50
150	100	<i>Lipton66</i>	with		0.99	29.6	9.13	1.03	3.39	12.5	0.97	33.6	18.9	2.21	2.09
150	100	<i>Lipton61</i>	with		<u>1.00</u>	<u>33.7</u>	<u>13.5</u>	<u>1.65</u>	<u>4.33</u>	<u>8.81</u>	<u>1.37</u>	<u>33.3</u>	<u>19.8</u>	<u>2.85</u>	<u>2.70</u>
MEAN					1.02	32.2	13.1	1.46	3.66	11.5	1.11	32.5	20.7	2.40	2.46
150	100	<i>Lipton</i>	with		1.09	33.7	10.7	1.14	4.56	13.0	0.85	34.8	20.5	1.95	2.05
150	100	<i>Lipton</i>	with		0.95	32.6	14.3	1.32	4.12	15.3	0.87	35.1	21.9	2.41	2.45
150	100	<i>Lipton</i>	with		0.89	29.7	11.1	1.21	3.68	9.70	0.79	29.9	16.3	1.96	2.54
150	100	<i>Lipton</i>	with		<u>0.92</u>	<u>33.4</u>	<u>8.53</u>	<u>1.13</u>	<u>2.61</u>	<u>8.21</u>	<u>1.48</u>	<u>28.5</u>	<u>17.4</u>	<u>2.29</u>	<u>1.96</u>
MEAN					0.98	32.4	11.2	1.20	3.75	11.6	1.00	32.1	19.0	2.15	2.25
150	200	<i>BrisolA2</i>	with		0.96	29.9	12.1	1.23	10.3	5.21	1.17	27.8	22.2	2.14	5.81
150	200	<i>BrisolD6</i>	with		0.96	35.1	11.7	1.28	3.01	10.6	0.79	30.6	20.6	2.28	1.83
150	200	<i>BrisolB3</i>	with		1.02	33.8	11.6	1.78	3.21	11.5	0.88	28.8	17.9	3.17	1.75
150	200	<i>BrisolD8</i>	with		<u>1.48</u>	<u>41.9</u>	<u>9.72</u>	<u>1.37</u>	<u>5.19</u>	<u>9.81</u>	<u>0.96</u>	<u>28.3</u>	<u>15.0</u>	<u>2.10</u>	<u>1.61</u>
MEAN					1.11	35.2	11.3	1.42	5.42	9.29	0.95	28.9	18.9	2.42	2.75
150	200	<i>Bristol</i>	with		0.87	30.9	11.0	1.17	1.85	9.33	0.91	26.6	16.3	1.56	1.44
150	200	<i>Bristol</i>	with		1.01	34.6	15.5	1.49	2.92	8.00	0.92	27.1	15.3	1.57	1.40
150	200	<i>Bristol</i>	with		0.94	35.4	9.87	1.29	2.97	8.49	0.76	25.0	19.9	2.06	3.92
150	200	<i>Bristol</i>	with		<u>0.96</u>	<u>32.1</u>	<u>11.5</u>	<u>1.24</u>	<u>3.70</u>	<u>10.6</u>	<u>1.00</u>	<u>28.9</u>	<u>18.8</u>	<u>2.26</u>	<u>2.00</u>
MEAN					0.95	33.2	12.0	1.30	2.86	9.10	0.90	26.9	17.6	1.86	2.19

Table A.12: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Straw				Pod walls							
				S	P	K	Ca	Mg	Cl	S	P	K	Ca	Mg	Cl
2002/2003															
150	200	<i>Lipton</i>	01	with	0.85	33.0	9.12	1.03	2.78	10.1	0.66	34.2	15.7	2.30	2.09
150	200	<i>Lipton</i>	43	with	0.84	33.1	11.6	1.44	4.64	9.26	0.78	32.7	15.4	1.81	2.76
150	200	<i>Lipton</i>	31	with	1.07	32.1	14.8	1.85	4.14	9.09	0.91	31.3	15.7	1.90	1.69
150	200	<i>Lipton</i>	24	with	0.91	28.3	13.3	1.35	3.94	13.3	1.21	28.9	24.3	2.29	3.28
MEAN					0.92	31.6	12.2	1.42	3.88	10.4	0.89	31.8	17.8	2.08	2.45
150	200	<i>Lipton</i>		without	0.93	34.8	9.58	1.10	2.83	8.98	0.83	34.4	23.5	2.10	2.75
150	200	<i>Lipton</i>		without	0.77	31.1	11.6	1.13	2.98	11.5	1.00	34.0	17.0	2.02	2.52
150	200	<i>Lipton</i>		without	1.21	34.9	15.9	1.50	3.14	11.3	1.18	34.4	19.2	2.52	2.25
150	200	<i>Lipton</i>		without	0.96	34.3	11.3	1.39	3.10	9.60	1.25	34.9	16.8	2.18	1.90
MEAN					5.42	33.8	12.1	1.28	3.03		1.07	34.4	19.1	2.21	2.35

Table A.13: Organic sulphur compounds in leaf and seed samples, Braunschweig, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				Leaves				Seeds						
				γ -glu-cys	GSH	Prog	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GNF	GSL		
$\mu\text{mol g}^{-1}$																		
2000/2001																		
0	100	Bristol	with	0.99	1.07	10.6	0.27	2.06	0.54	0.08	0.17	3.39	2.79	2.83	0.60	1.49	0.26	5.18
0	100	Bristol	with	1.03	0.94	7.36	0.13	1.96	0.45	0.07	n.d.	2.81	3.06	3.07	0.63	1.84	0.17	5.96
0	100	Bristol	with	1.12	0.90	8.23	0.27	3.37	0.76	0.14	n.d.	4.83	2.96	3.69	0.76	2.31	0.25	7.17
0	100	Bristol	with	—	0.74	16.7	0.34	1.08	0.39	0.07	n.d.	2.00	2.35	3.22	0.86	1.86	0.13	6.12
MEAN				0.93	0.91	10.7	0.26	2.12	0.54	0.09	0.17	3.26	2.79	3.20	0.71	1.87	0.20	6.11
0	100	Bristol	without	1.01	0.93	11.6	0.93	1.57	0.63	0.08	0.26	3.76	3.44	3.78	0.71	2.26	0.29	7.37
0	100	Bristol	without	0.96	0.88	10.4	0.28	2.62	0.70	0.10	n.d.	3.93	3.44	3.44	0.67	2.17	0.19	7.08
0	100	Bristol	without	1.26	0.66	16.8	0.25	1.78	0.33	0.08	n.d.	2.65	2.49	3.08	0.60	1.88	0.22	6.08
0	100	Bristol	without	—	1.16	9.23	0.55	2.26	0.46	0.13	n.d.	3.68	2.28	3.92	0.90	2.86	0.27	8.16
MEAN				1.05	0.91	12.0	0.50	2.06	0.53	0.10	0.26	3.51	2.91	3.55	0.72	2.29	0.24	7.17
0	100	Lipton	with	0.72	1.04	10.9	0.72	2.45	0.86	0.09	n.d.	4.35	3.19	4.71	0.94	2.33	0.28	8.65
0	100	Lipton	with	0.92	0.89	8.02	0.73	2.18	0.85	0.08	0.05	4.09	3.31	3.95	0.97	1.99	0.28	7.38
0	100	Lipton	with	0.91	0.89	18.3	0.37	1.92	0.58	0.07	0.04	3.16	1.41	4.26	0.87	2.19	0.15	8.18
0	100	Lipton	with	—	0.96	8.56	1.50	2.28	0.74	0.10	0.05	4.92	2.84	4.47	0.94	2.17	0.20	8.21
MEAN				0.87	0.95	11.4	0.83	2.21	0.76	0.08	0.05	4.13	2.69	4.34	0.93	2.17	0.23	8.11
0	100	Lipton	without	0.64	0.99	9.15	0.75	2.77	0.98	0.13	0.04	4.91	2.43	3.80	0.63	1.71	0.19	6.32
0	100	Lipton	without	1.29	0.92	8.32	0.37	1.40	0.40	0.06	0.04	2.39	2.56	3.85	0.73	1.64	0.20	6.81
0	100	Lipton	without	1.06	0.92	9.82	0.54	1.75	0.59	0.09	n.d.	3.12	2.83	4.55	1.03	2.00	n.d.	7.83
0	100	Lipton	without	—	1.07	18.6	0.35	1.68	0.50	0.08	0.07	2.88	2.11	4.29	0.71	1.89	0.25	7.23
MEAN				0.85	0.97	11.5	0.50	1.90	0.61	0.09	0.05	3.32	2.48	4.12	0.77	1.81	0.21	7.05
0	200	Bristol	with	0.84	1.01	17.3	n.d.	1.45	0.32	0.02	0.16	2.37	2.40	3.04	0.42	1.84	0.15	6.04
0	200	Bristol	with	0.87	1.01	7.84	0.38	1.74	0.52	0.10	n.d.	2.92	2.67	3.63	0.55	2.13	0.20	6.53
0	200	Bristol	with	1.09	0.85	11.4	0.29	1.59	0.54	0.12	n.d.	2.86	2.67	4.05	0.96	2.56	0.31	7.88
0	200	Bristol	with	—	0.85	15.1	0.09	2.02	0.45	0.06	0.05	2.83	2.39	3.47	0.64	2.12	0.14	7.07
MEAN				0.78	0.93	12.9	0.25	1.70	0.46	0.08	0.11	2.74	2.53	3.55	0.64	2.16	0.20	6.88
0	200	Bristol	without	0.91	1.15	10.7	0.52	2.14	0.65	0.08	0.26	3.90	2.85	3.59	0.57	2.16	0.22	6.87
0	200	Bristol	without	0.80	1.07	8.20	0.22	2.14	0.45	0.10	0.05	3.18	2.60	3.46	0.61	2.00	0.21	6.57
0	200	Bristol	without	0.89	0.90	17.7	0.27	2.01	0.51	0.07	n.d.	3.05	2.02	3.35	0.57	2.08	0.20	6.12
0	200	Bristol	without	—	1.11	13.4	0.35	2.39	0.42	0.11	n.d.	3.64	2.04	3.26	0.63	2.24	0.35	6.49
MEAN				0.77	1.06	12.5	0.34	2.17	0.51	0.09	0.16	3.44	2.38	3.42	0.60	2.12	0.25	6.51

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GNF	GSL
$\mu\text{mol g}^{-1}$																		
<i>2000/2001</i>																		
0	200	<i>Lipton</i>	with	0.63	0.98	9.70	0.46	1.62	0.57	0.07	0.05	2.95	5.33	5.09	0.72	3.06	0.20	9.84
0	200	<i>Lipton</i>	with	0.73	0.98	17.3	0.31	1.14	0.35	0.05	0.04	2.03	2.83	5.08	0.94	2.24	0.17	8.91
0	200	<i>Lipton</i>	with	0.79	1.04	8.16	0.67	1.42	0.55	0.07	0.00	2.85	2.59	4.77	0.89	2.42	0.16	8.29
0	200	<i>Lip0205</i>	with	—	0.79	17.0	0.48	1.73	0.56	0.06	0.04	3.05	2.04	3.14	0.42	1.58	0.15	5.29
MEAN					0.95	13.0	0.48	1.48	0.51	0.06	0.03	2.72	3.20	4.52	0.74	2.32	0.17	8.08
0	200	<i>Lipton</i>	without	0.66	1.16	8.42	0.43	1.74	0.60	0.09	0.05	3.05	2.89	5.11	0.70	2.62	0.22	9.09
0	200	<i>Lipton</i>	without	0.80	0.76	16.7	0.40	1.18	0.40	0.05	0.04	2.19	1.85	3.92	0.53	1.62	0.26	6.70
0	200	<i>Lipton</i>	without	0.74	1.00	9.46	0.38	1.71	0.61	0.07	n.d.	2.91	2.42	4.61	0.78	2.17	0.20	7.79
0	200	<i>Lipton</i>	without	—	0.81	16.2	0.13	1.37	0.29	0.05	0.08	2.06	2.17	3.71	0.51	1.72	0.22	6.49
MEAN					0.93	12.7	0.33	1.50	0.47	0.07	0.06	2.55	2.33	4.34	0.63	2.03	0.22	7.52
150	100	<i>Bristol</i>	with	2.46	3.31	23.5	0.27	1.59	0.57	0.06	0.20	3.23	5.87	4.73	1.32	2.85	0.44	10.0
150	100	<i>Bristol</i>	with	1.82	2.68	7.17	0.48	2.82	0.80	0.13	0.08	4.59	4.57	5.29	1.03	3.67	0.36	10.3
150	100	<i>Bristol</i>	with	1.65	1.76	7.66	0.75	3.71	0.78	0.26	n.d.	5.91	1.76	4.70	1.74	2.62	0.51	9.58
150	100	<i>Bristol04</i>	with	—	2.40	7.08	0.45	2.47	0.66	0.10	0.05	3.99	2.38	4.28	1.13	2.63	0.24	8.56
MEAN					2.54	11.4	0.49	2.65	0.70	0.14	0.11	4.43	3.65	4.75	1.31	2.94	0.39	9.61
150	100	<i>Bristol</i>	without	2.43	3.79	6.95	0.50	2.88	1.23	0.10	0.48	5.44	5.85	5.09	1.11	3.06	0.41	9.89
150	100	<i>Bristol</i>	without	1.55	2.16	7.42	0.40	2.43	0.65	0.16	0.09	3.95	5.04	5.35	1.08	3.14	0.23	10.1
150	100	<i>Bristol</i>	without	2.52	2.50	7.43	0.42	2.23	0.53	0.10	0.04	3.57	5.98	4.44	0.84	2.79	0.22	8.64
150	100	<i>Bristol</i>	without	—	2.66	8.24	0.43	2.99	0.70	0.21	0.07	4.72	0.13	5.49	1.54	3.01	0.40	11.2
MEAN					2.78	7.51	0.44	2.63	0.78	0.14	0.17	4.42	4.25	5.09	1.14	3.00	0.31	9.97
150	100	<i>Lipton</i>	with	1.54	2.63	22.7	2.30	2.36	1.82	0.22	0.25	7.50	3.31	5.51	1.11	2.70	0.27	10.3
150	100	<i>Lipton</i>	with	1.54	1.56	13.7	0.84	2.61	0.98	0.13	0.04	4.83	5.48	5.70	1.45	2.75	0.25	10.6
150	100	<i>Lipton</i>	with	1.87	1.62	22.2	1.52	2.70	1.05	0.12	n.d.	5.61	1.87	6.19	1.39	3.16	0.27	11.3
150	100	<i>Lipton04</i>	with	—	1.72	19.8	0.45	1.80	0.70	0.08	0.17	3.33	1.09	4.92	1.41	2.00	0.24	9.14
MEAN					1.88	19.6	1.28	2.37	1.14	0.14	0.16	5.32	2.94	5.58	1.34	2.65	0.26	10.3
150	100	<i>Lipton</i>	without	1.54	1.80	22.3	0.82	2.37	0.96	0.13	0.05	4.51	3.88	6.43	1.16	3.16	0.32	11.5
150	100	<i>Lipton</i>	without	1.55	1.20	13.1	0.88	2.39	0.85	0.11	0.05	4.48	5.59	5.89	1.29	2.78	0.28	10.6
150	100	<i>Lipton</i>	without	1.45	1.51	8.40	1.01	2.53	1.02	0.10	n.d.	4.90	2.55	5.43	1.38	2.45	0.30	9.56
150	100	<i>Lipton</i>	without	—	2.32	13.9	0.62	2.28	0.81	0.10	0.07	4.07	2.27	5.82	1.31	2.73	0.23	10.7
MEAN					1.71	14.4	0.83	2.39	0.91	0.11	0.06	4.49	3.57	5.89	1.29	2.78	0.28	10.6

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GNF	GSL
2000/2001																		
150	200	<i>Bristol</i>	with	3.09	3.30	22.6	0.16	2.51	0.85	0.04	0.23	4.62	4.90	m.v.	m.v.	m.v.	m.v.	
150	200	<i>Bristol</i>	with	2.88	2.78	15.4	0.35	2.02	0.55	0.10	0.06	3.28	7.10	5.02	1.21	3.17	0.31	10.4
150	200	<i>Bristol</i>	with	2.62	2.76	14.9	0.75	2.95	0.74	0.15	n.d.	4.88	3.63	4.74	1.09	2.96	0.34	9.21
150	200	<i>Bristol</i>	with	—	2.29	8.65	0.35	2.19	0.63	0.10	0.07	3.52	1.63	5.47	1.24	3.23	0.23	10.9
MEAN				2.88	2.78	15.4	0.40	2.42	0.69	0.10	0.12	4.07	4.32	5.08	1.18	3.12	0.29	10.2
150	200	<i>Bristol</i>	without	3.36	3.24	8.80	0.28	2.15	0.80	0.06	0.28	4.43	5.67	5.27	0.98	3.34	0.29	10.5
150	200	<i>Bristol</i>	without	2.09	2.28	21.6	0.47	2.59	0.50	0.07	0.07	4.04	5.45	4.46	0.97	2.94	0.19	9.07
150	200	<i>Bristol</i>	without	2.07	2.17	6.92	0.55	2.68	0.70	0.17	n.d.	4.42	6.24	5.78	1.21	3.30	0.36	10.7
150	200	<i>Bristol</i>	without	—	2.06	6.55	0.48	2.77	0.68	0.13	n.d.	4.32	3.07	4.97	1.14	3.23	0.23	9.68
MEAN				2.51	2.44	11.0	0.44	2.55	0.67	0.11	0.17	4.30	5.11	5.12	1.08	3.20	0.27	9.99
150	200	<i>Lipton</i>	with	2.67	4.19	24.4	2.64	2.09	1.25	0.14	0.23	6.62	3.92	6.49	0.79	2.84	0.25	10.6
150	200	<i>Lipton</i>	with	1.76	2.34	21.4	0.58	1.92	0.78	0.16	0.06	3.66	5.94	6.64	1.32	2.96	0.20	11.3
150	200	<i>Lipton</i>	with	2.15	2.26	23.7	0.75	2.57	0.87	0.13	n.d.	4.54	1.45	6.15	1.17	3.11	0.28	10.9
150	200	<i>Lipton</i>	with	—	1.98	23.5	0.51	2.28	0.76	0.13	0.05	3.90	2.27	6.17	1.33	3.03	0.26	10.8
MEAN				2.19	2.69	23.2	1.12	2.21	0.91	0.14	0.11	4.68	3.39	6.36	1.15	2.98	0.25	10.9
150	200	<i>Lipton</i>	without	2.27	3.41	17.2	2.78	2.51	1.55	0.20	0.28	7.53	5.82	6.83	0.87	3.58	0.31	12.0
150	200	<i>Lipton</i>	without	2.63	3.01	22.3	0.65	2.16	0.74	0.11	0.05	3.90	3.25	6.89	1.43	3.54	0.23	12.4
150	200	<i>Lipton</i>	without	1.38	1.43	15.6	0.61	2.14	0.74	0.16	0.04	3.89	3.94	m.v.	m.v.	m.v.	m.v.	m.v.
150	200	<i>Lipton</i>	without	—	1.30	22.2	0.58	2.13	0.76	0.12	0.07	3.86	2.30	6.25	1.21	2.78	0.23	11.0
MEAN				1.98	2.29	19.3	1.15	2.24	0.95	0.15	0.11	4.80	3.83	6.66	1.17	3.30	0.26	11.8
2001/2002																		
S rate N rate Variety Fungicide				Leaves										Seeds				
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
2001/2002																		
0	100	<i>Bristol</i>	with	—	0.58	9.86	0.25	2.31	0.39	0.11	0.11	4.12	3.79	4.83	0.90	2.21	0.07	9.17
0	100	<i>Bristol</i>	with	—	0.57	28.7	0.32	1.98	0.35	0.11	0.11	3.38	2.20	3.95	0.71	1.65	0.05	7.24
0	100	<i>Bristol</i>	with	—	0.36	24.8	0.52	2.12	0.49	0.08	0.08	3.42	2.39	4.93	0.86	2.12	0.03	9.50
0	100	<i>Bristol</i>	with	—	0.52	32.8	1.63	2.99	0.55	0.14	0.14	5.73	1.87	5.00	0.09	2.17	n.d.	9.78
MEAN				1.58	0.51	24.0	0.68	2.35	0.44	0.11	0.11	4.16	2.56	4.68	0.64	2.04	0.05	8.92

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
2001/2002																		
μmol g ⁻¹																		
0	100	Bristol	without	1.70	0.54	10.2	0.64	3.29	0.85	0.14	0.14	5.41	3.74	5.16	0.88	2.58	0.02	10.0
0	100	Bristol	with		0.55	28.5	0.41	2.40	0.53	0.09	0.09	3.80	2.65	4.49	0.79	2.06	0.09	8.56
0	100	Bristol	without		0.44	26.1	2.71	5.21	1.16	0.15	0.15	10.7	2.50	5.16	0.70	1.85	0.06	9.49
0	100	Bristol	with		0.28	19.4	1.63	3.24	0.71	0.12	0.12	5.97	3.57	3.86	0.83	1.67	0.02	8.72
MEAN					0.45	21.0	1.35	3.53	0.81	0.13	0.13	6.48	3.12	4.67	0.80	2.04	0.05	9.20
0	100	Lipton40	with		0.52	10.4	1.10	2.70	0.41	0.11	0.11	4.91	3.98	6.23	1.08	2.43	0.02	11.1
0	100	Lipton42	with		0.53	10.8	1.45	3.26	0.59	0.17	0.17	5.97	4.17	5.23	0.99	2.14	0.14	9.77
0	100	Lipton49	with		0.59	38.9	0.48	2.71	0.44	0.05	0.05	3.93	2.33	6.52	1.49	2.34	0.04	13.1
0	100	Lipton43	with		0.56	30.7	1.84	3.94	0.73	0.15	0.15	6.94	2.72	7.55	0.43	2.80	0.05	13.5
MEAN					0.55	22.7	1.22	3.15	0.54	0.12	0.12	5.43	3.30	6.38	1.00	2.43	0.06	11.8
0	100	Lipton	without		0.87	11.7	1.25	2.83	0.64	0.08	0.08	5.29	3.25	5.11	0.87	2.07	0.07	9.24
0	100	Lipton	with		0.92	14.2	1.77	4.23	0.86	0.23	0.23	7.73	2.58	5.55	0.98	2.32	0.09	9.81
0	100	Lipton	without		0.65	29.0	0.30	1.66	0.34	0.03	0.03	2.42	2.47	6.53	0.95	2.30	n.d.	11.7
0	100	Lipton	with		0.79	37.3	1.02	2.20	0.46	0.11	0.11	4.15	2.13	6.41	0.27	2.35	0.04	11.4
MEAN					0.81	23.1	1.08	2.73	0.58	0.11	0.11	4.90	2.61	5.90	0.77	2.26	0.07	10.5
0	200	Bristol64	with		0.76	22.6	0.64	3.01	0.74	0.08	0.08	4.97	2.60	4.81	0.77	2.32	0.02	8.70
0	200	Bristol61	with		0.25	23.5	0.33	1.47	0.30	0.05	0.05	2.62	2.71	4.53	0.92	2.24	0.06	12.7
0	200	Bristol64	with		0.41	29.2	1.92	5.94	1.68	m.v.	m.v.	10.4	2.19	5.16	0.76	1.90	0.20	9.30
0	200	Bristol65	with		0.45	29.6	0.64	3.39	0.77	0.12	0.12	5.27	2.09	5.02	0.45	2.42	0.03	10.0
MEAN					0.47	26.2	0.88	3.46	0.87	0.09	0.09	5.81	2.40	4.88	0.72	2.22	0.08	10.2
0	200	Bristol	without		0.56	12.5	1.20	5.50	1.42	0.28	0.28	9.66	4.09	5.69	0.90	2.21	0.04	10.0
0	200	Bristol	with		0.49	28.4	0.34	2.25	0.45	0.12	0.12	3.86	2.30	4.07	0.67	1.73	0.07	7.65
0	200	Bristol	without		0.92	36.7	1.44	2.71	0.62	0.14	0.09	5.07	2.59	5.07	0.95	2.07	0.19	9.65
0	200	Bristol	with		0.50	26.8	0.61	3.25	0.64	0.08	0.08	4.93	2.94	5.14	n.d.	2.09	n.d.	11.9
MEAN					0.62	26.1	0.90	3.43	0.79	0.15	0.15	5.88	2.98	4.99	0.84	2.03	0.10	9.79
0	200	Lipton60	with		0.90	15.7	1.57	3.38	0.61	0.14	0.32	6.22	3.61	5.66	0.84	2.34	0.07	10.1
0	200	Lipton63	with		0.82	10.8	1.21	2.66	0.58	0.20	0.30	5.03	3.15	4.43	0.80	1.70	0.10	7.97
0	200	Lipton1	with		0.61	10.0	2.72	5.29	1.19	0.28	0.20	10.0	2.75	5.93	3.61	2.00	0.19	13.3
0	200	Lipton1	with		0.53	34.7	0.40	1.04	0.25	0.17	0.17	1.89	2.20	5.36	0.32	1.90	0.04	9.62
MEAN					0.72	17.8	1.48	3.09	0.66	0.16	0.25	5.79	2.93	5.34	1.39	1.98	0.10	10.2

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
2001/2002																		
0	200	Lipton	without	1.72	1.68	15.7	0.68	1.74	0.41	0.12	0.38	3.54	2.81	5.99	0.95	2.32	0.05	10.5
0	200	Lipton	with	—	1.34	15.6	1.03	2.43	0.53	0.15	0.28	4.58	3.70	6.45	n.d.	2.70	n.d.	11.3
0	200	Lipton	without	—	0.76	36.5	1.36	3.82	1.02	0.09	0.13	6.57	2.39	5.54	0.43	1.82	0.06	10.2
0	200	Lipton	without	—	<u>0.67</u>	<u>36.4</u>	<u>1.63</u>	<u>3.24</u>	<u>0.71</u>	<u>0.09</u>	<u>0.07</u>	<u>5.97</u>	<u>2.43</u>	<u>5.92</u>	<u>0.27</u>	<u>2.34</u>	<u>0.04</u>	<u>10.6</u>
MEAN				—	1.11	26.0	1.17	2.81	0.67	0.12	0.22	5.16	2.84	5.97	0.55	2.29	0.05	10.6
150	100	Bristol73	with	—	0.63	22.8	0.39	1.68	0.34	0.05	0.33	2.90	7.02	8.17	1.75	3.59	0.06	15.1
150	100	Bristol65	with	—	0.70	11.7	0.72	3.87	0.72	0.13	0.33	5.88	4.16	7.11	1.68	3.14	0.06	13.1
150	100	Bristol81	with	—	1.50	12.7	1.44	4.61	0.94	0.17	0.10	7.42	6.21	7.22	1.64	3.42	0.03	15.4
150	100	Bristol60	with	—	<u>1.39</u>	<u>40.3</u>	<u>1.66</u>	<u>2.94</u>	<u>0.66</u>	<u>0.13</u>	<u>0.19</u>	<u>5.82</u>	<u>2.45</u>	<u>7.58</u>	<u>m.v.</u>	<u>3.30</u>	<u>n.d.</u>	<u>13.8</u>
MEAN				—	1.05	21.9	1.05	3.27	0.67	0.12	0.24	5.50	4.96	7.52	1.69	3.36	0.05	14.4
150	100	Bristol	without	—	0.97	15.3	1.62	4.88	1.20	0.22	0.34	8.39	6.14	8.25	1.73	3.98	0.03	15.7
150	100	Bristol	with	—	0.96	15.7	0.96	5.01	1.06	0.38	m.v.	9.59	4.05	7.08	1.45	3.48	0.07	13.5
150	100	Bristol	without	—	1.22	43.9	0.91	2.06	0.33	0.07	0.06	3.77	5.01	7.00	1.35	3.71	0.03	14.0
150	100	Bristol	without	—	<u>1.93</u>	<u>49.8</u>	<u>0.74</u>	<u>2.90</u>	<u>0.67</u>	<u>0.12</u>	<u>0.17</u>	<u>4.76</u>	<u>5.27</u>	<u>6.27</u>	<u>0.36</u>	<u>3.24</u>	<u>0.05</u>	<u>12.8</u>
MEAN				—	1.27	31.2	1.06	3.71	0.82	0.20	0.19	6.63	5.12	7.15	1.22	3.60	0.05	14.0
150	100	Lipton62	with	—	0.90	12.6	2.11	5.02	0.99	0.20	0.38	8.81	6.57	8.28	1.72	3.26	0.03	14.7
150	100	Lipton65	with	—	1.21	14.2	3.66	8.45	1.55	0.42	0.64	14.9	3.20	7.49	1.79	3.31	0.08	14.1
150	100	Lipton97	with	—	0.74	37.9	1.51	3.28	0.75	0.32	0.29	6.34	6.47	6.61	1.30	2.83	n.d.	12.2
150	100	Lipton60	with	—	<u>1.71</u>	<u>12.9</u>	<u>0.40</u>	<u>1.91</u>	<u>0.49</u>	<u>0.04</u>	<u>0.22</u>	<u>3.20</u>	<u>3.21</u>	<u>7.67</u>	<u>0.23</u>	<u>2.86</u>	<u>0.04</u>	<u>14.2</u>
MEAN				—	1.14	19.4	1.92	4.66	0.95	0.24	0.38	8.32	4.86	7.51	1.26	3.06	0.05	13.8
150	100	Lipton	without	—	1.03	26.4	2.43	4.74	1.06	0.23	0.37	9.08	6.97	8.74	1.84	3.68	0.02	15.6
150	100	Lipton	without	—	0.74	12.4	2.81	6.04	1.22	0.42	0.57	11.4	2.02	7.07	1.47	3.29	0.05	13.1
150	100	Lipton	without	—	1.31	11.6	0.82	2.15	0.47	0.17	0.15	3.78	5.84	8.34	1.56	3.14	0.36	15.2
150	100	Lipton	without	—	<u>0.92</u>	<u>42.6</u>	<u>0.37</u>	<u>2.87</u>	<u>0.40</u>	<u>0.05</u>	<u>0.04</u>	<u>3.95</u>	<u>7.24</u>	<u>8.88</u>	<u>0.18</u>	<u>3.43</u>	<u>n.d.</u>	<u>15.6</u>
MEAN				—	1.00	23.2	1.61	3.95	0.79	0.22	0.28	7.05	5.52	8.26	1.26	3.38	0.15	14.9
150	200	Bristol29	with	—	1.09	27.1	0.80	3.38	0.81	0.09	0.29	5.70	7.17	12.5	2.49	4.79	0.55	22.4
150	200	Bristol78	with	—	0.52	17.4	0.81	3.08	0.67	0.11	0.27	5.07	8.35	7.99	1.60	3.83	0.06	14.5
150	200	Bristol84	with	—	1.22	12.2	0.07	2.56	0.64	0.10	0.13	3.55	5.09	9.26	1.00	4.03	0.03	17.5
150	200	Bristol92	with	—	<u>1.32</u>	<u>12.1</u>	<u>0.48</u>	<u>2.21</u>	<u>0.55</u>	<u>0.03</u>	<u>0.23</u>	<u>3.61</u>	<u>6.19</u>	<u>9.23</u>	<u>0.26</u>	<u>4.35</u>	<u>0.06</u>	<u>17.2</u>
MEAN				—	1.04	17.2	0.54	2.81	0.67	0.08	0.23	4.48	6.70	9.74	1.34	4.25	0.17	17.9

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
2001/2002																		
150	200	<i>Bristol</i>	without	3.11	1.05	28.6	1.24	3.93	1.19	0.18	0.29	6.91	7.00	8.60	1.60	3.46	0.35	15.5
150	200	<i>Bristol</i>	without	1.89	0.75	12.4	0.34	1.60	0.35	0.09	0.30	2.77	6.05	7.93	1.46	3.89	0.07	14.5
150	200	<i>Bristol</i>	without	2.87	0.88	16.2	0.36	2.07	0.49	0.06	0.17	3.22	6.38	9.95	1.81	3.89	0.05	20.9
150	200	<i>Bristol</i>	with	—	<u>1.66</u>	<u>15.9</u>	<u>1.80</u>	<u>3.17</u>	<u>0.67</u>	<u>0.01</u>	<u>0.28</u>	<u>6.11</u>	<u>5.59</u>	<u>8.65</u>	<u>0.13</u>	<u>4.14</u>	<u>0.03</u>	<u>16.3</u>
MEAN				2.69	1.09	18.3	0.94	2.69	0.67	0.09	0.26	4.75	6.25	8.78	1.25	3.84	0.13	16.8
150	200	<i>Lipton</i>	with	2.68	1.02	17.5	1.87	3.58	0.69	0.20	0.39	6.92	8.45	8.78	1.36	3.32	0.06	14.9
150	200	<i>Lipton</i>	with	2.79	1.02	21.9	2.07	3.67	0.77	0.20	0.34	7.22	5.14	9.12	1.77	3.68	0.02	15.9
150	200	<i>Lipton</i>	with	2.08	0.68	11.4	1.64	2.78	0.69	0.19	0.30	5.73	7.27	8.70	0.31	3.47	0.37	13.7
150	200	<i>Lipton</i> 48	with	—	<u>2.19</u>	<u>18.3</u>	<u>0.47</u>	<u>2.25</u>	<u>0.47</u>	<u>0.05</u>	<u>0.19</u>	<u>3.51</u>	<u>6.74</u>	<u>9.57</u>	<u>0.28</u>	<u>3.40</u>	<u>0.04</u>	<u>16.9</u>
MEAN				2.76	1.23	17.3	1.51	3.07	0.66	0.16	0.31	5.85	6.90	9.04	0.93	3.47	0.12	15.4
150	200	<i>Lipton</i>	without	2.42	1.38	14.5	1.42	2.77	0.64	0.23	0.39	5.65	6.82	9.74	1.80	3.71	0.06	17.0
150	200	<i>Lipton</i>	without	2.16	1.13	14.7	1.63	3.13	0.66	0.27	0.52	6.36	5.66	7.76	1.32	3.22	0.09	13.4
150	200	<i>Lipton</i>	without	2.33	1.07	11.8	1.11	2.27	0.44	0.19	0.31	4.46	7.83	9.12	0.24	3.61	0.34	14.8
150	200	<i>Lipton</i>	with	—	<u>2.17</u>	<u>16.5</u>	<u>0.04</u>	<u>1.93</u>	<u>0.44</u>	<u>0.06</u>	<u>0.12</u>	<u>2.58</u>	<u>6.97</u>	<u>9.54</u>	<u>0.24</u>	<u>3.48</u>	<u>0.02</u>	<u>16.6</u>
MEAN				2.49	1.44	14.4	1.05	2.52	0.54	0.19	0.33	4.76	6.82	9.04	0.90	3.51	0.13	15.4
S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cy s	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL

2002/2003

2002/2003																		
0	100	<i>Bristol</i> 48	with	—	0.43	10.6	0.09	0.36	0.16	0.05	n.d.	0.66	2.10	2.36	0.11	1.33	n.d.	4.17
0	100	<i>Bristol</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	1.62	0.09	0.79	0.02	2.76
0	100	<i>Bristol</i>	with	0.86	0.60	14.6	0.08	0.71	0.22	0.11	n.d.	1.11	2.18	2.37	0.11	0.93	0.02	3.58
0	100	<i>Bristol</i> 39	with	—	<u>0.92</u>	<u>16.0</u>	<u>0.32</u>	<u>0.91</u>	<u>0.35</u>	<u>0.32</u>	<u>0.06</u>	<u>1.96</u>	<u>2.42</u>	<u>3.05</u>	<u>0.38</u>	<u>1.40</u>	<u>0.04</u>	<u>5.24</u>
MEAN				0.58	0.65	13.7	0.16	0.66	0.24	0.16	0.06	1.24	2.23	2.35	0.17	1.11	0.03	3.94
0	100	<i>Bristol</i>	with	0.89	0.70	15.0	n.d.	0.54	0.22	0.10	n.d.	0.93	2.03	1.64	0.08	0.72	n.d.	2.77
0	100	<i>Bristol</i>	without	n.a.	0.60	15.2	0.12	1.21	0.32	0.12	n.d.	1.81	2.03	2.05	0.20	0.81	0.02	3.26
0	100	<i>Bristol</i>	without	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	1.94	0.11	0.96	0.02	3.52
0	100	<i>Bristol</i>	with	—	<u>0.68</u>	<u>15.3</u>	<u>n.d.</u>	<u>0.38</u>	<u>0.19</u>	<u>0.09</u>	<u>n.d.</u>	<u>0.67</u>	<u>2.15</u>	<u>1.98</u>	<u>0.14</u>	<u>1.21</u>	<u>0.02</u>	<u>3.67</u>
MEAN				0.82	0.66	15.1	0.12	0.71	0.24	0.11	n.d.	1.14	2.07	1.90	0.13	0.92	0.02	3.31

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds						
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prot-S	Prog	GBN	GN	GB	GSL	
				μmol g ⁻¹																
				2002/2003																
0	100	Lipton	with	0.71	0.53	13.4	0.31	0.86	0.26	0.10	n.d.	1.61	1.84	3.87	0.18	1.74	n.d.	6.05		
0	100	Lipton	with	0.69	0.41	10.8	0.43	0.82	0.28	0.13	n.d.	1.74	1.75	5.34	0.54	1.89	0.01	8.06		
0	100	Lipton	with	n.a.	0.57	15.0	0.27	0.68	0.21	0.10	0.09	1.52	1.99	3.27	0.31	1.54	0.02	5.62		
MEAN				0.80	0.50	13.1	0.34	0.79	0.25	0.11	0.09	1.63	1.86	4.16	0.32	1.67	0.02	6.50		
0	100	Lipton	without	n.a.	0.58	14.6	0.45	0.93	0.27	0.12	0.07	2.01	2.59	4.49	0.14	1.57	n.d.	6.51		
0	100	Lipton	without	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	5.21	0.39	2.03	n.d.	7.87		
0	100	Lipton	without	n.a.	0.78	20.0	0.46	1.08	0.28	0.17	n.d.	1.99	2.24	5.17	0.34	1.69	0.02	7.56		
0	100	Lipton	without	n.a.	0.25	3.62	1.27	0.88	0.35	0.29	0.14	2.10	2.59	4.45	0.30	1.54	0.02	6.52		
MEAN				0.46	0.53	12.7	0.73	0.96	0.30	0.19	0.11	2.03	2.47	4.83	0.29	1.71	0.02	7.12		
0	200	Bristol	with	n.a.	0.64	15.0	0.09	0.70	0.22	0.09	n.d.	1.10	2.63	1.19	0.11	0.50	0.05	1.98		
0	200	Bristol	with	n.a.	0.72	16.0	n.d.	0.20	0.10	0.05	n.d.	0.36	2.15	1.51	0.09	0.93	0.08	2.55		
0	200	Bristol	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	1.17	0.11	0.41	0.05	2.00		
0	200	Bristol	with	n.a.	0.73	17.1	n.d.	0.30	0.12	0.06	n.d.	0.48	1.78	1.36	0.16	0.57	0.05	2.60		
MEAN				0.70	0.70	16.0	0.09	0.40	0.15	0.07	n.d.	0.65	2.19	1.36	0.12	0.60	0.06	2.28		
0	200	Bristol	without	n.a.	0.78	15.8	0.08	0.31	0.14	0.06	n.d.	0.66	2.28	0.98	0.13	0.53	0.04	1.98		
0	200	Bristol	without	n.a.	0.58	16.0	0.11	0.90	0.26	0.12	n.d.	1.38	2.19	1.79	0.23	0.98	0.04	3.48		
0	200	Bristol	without	n.a.	0.70	17.2	0.12	0.54	0.23	0.10	n.d.	0.99	1.92	1.53	0.11	0.71	0.03	2.60		
0	200	Bristol	without	n.a.	0.45	10.3	0.45	0.70	0.23	0.13	n.d.	1.14	2.69	1.53	0.21	0.79	0.04	3.00		
MEAN				0.67	0.63	14.8	0.19	0.61	0.21	0.10	n.d.	1.04	2.27	1.53	0.17	0.75	0.04	2.77		
0	200	Lipton	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.62	0.15	1.66	0.02	5.67		
0	200	Lipton	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	4.50	0.27	0.92	0.06	7.58		
0	200	Lipton	with	0.51	0.46	11.4	0.44	0.83	0.28	0.11	0.06	1.72	2.00	3.66	0.26	1.07	0.08	5.31		
0	200	Lipton	with	n.a.	0.76	17.5	0.26	0.52	0.19	0.11	n.d.	1.08	1.78	3.83	0.23	1.70	0.05	5.87		
MEAN				0.51	0.61	14.4	0.35	0.68	0.24	0.11	0.06	1.40	1.89	3.83	0.23	1.34	0.05	6.11		
0	200	Lipton	without	0.49	0.89	17.0	0.27	0.49	0.18	0.17	0.07	1.19	1.21	4.87	0.23	2.52	0.05	7.83		
0	200	Lipton	without	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	4.06	0.19	1.88	0.07	6.57		
0	200	Lipton	without	0.68	0.66	17.7	0.64	1.18	0.44	0.37	0.14	2.50	1.38	4.11	0.22	1.25	0.05	5.86		
0	200	Lipton	without	n.a.	0.66	20.3	1.24	2.61	0.67	0.11	0.06	5.04	2.82	4.11	0.26	1.22	0.07	4.68		
MEAN				0.63	0.74	18.3	0.72	1.43	0.43	0.22	0.09	2.91	1.80	4.01	0.23	1.72	0.06	6.23		

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds						
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prot-S	Prog	GBN	GN	GB	GSL	
2002/2003																				
150	100	Bristol	with	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	6.35	0.36	2.50	0.06	9.51
150	100	Bristol	with	1.06	0.92	21.3	0.08	0.77	0.25	0.10	0.06	1.26	4.47	4.47	8.09	0.80	5.04	0.07	14.4	
150	100	Bristol	with	0.49	0.92	18.5	0.12	0.74	n.d.	0.13	n.d.	0.99	3.58	3.58	6.03	0.46	2.96	0.07	9.82	
150	100	Bristol	with	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	5.26	0.39	2.15	0.07	8.09	
MEAN				0.78	0.92	19.9	0.10	0.76	0.25	0.11	0.06	1.12	4.02	4.02	6.43	0.50	3.16	0.07	10.5	
150	100	Bristol	without	1.03	0.79	19.9	0.13	0.77	0.22	0.15	0.13	1.38	5.61	5.61	5.03	0.32	2.30	0.06	8.00	
150	100	Bristol	with	0.54	0.54	15.8	0.16	1.26	0.31	0.10	n.d.	1.89	3.07	3.07	5.67	0.46	2.00	0.08	8.37	
150	100	Bristol	with	1.01	1.01	20.4	0.14	0.54	0.27	0.09	n.d.	1.04	2.67	2.67	3.19	0.26	1.70	0.13	5.50	
150	100	Bristol	with	1.09	1.09	11.3	0.28	1.92	0.45	0.19	n.d.	2.84	6.19	6.19	4.75	0.53	2.67	0.05	8.52	
MEAN				0.99	0.86	16.9	0.18	1.12	0.31	0.13	0.13	1.79	4.38	4.38	4.66	0.39	2.17	0.08	7.60	
150	100	Lipton	with	0.96	0.69	8.35	0.43	1.06	0.32	0.13	0.06	2.05	4.93	4.93	7.54	0.48	2.61	0.05	10.9	
150	100	Lipton	with	0.85	0.81	20.9	0.73	1.57	0.48	0.21	0.11	3.13	4.64	4.64	8.23	0.52	3.74	0.08	12.8	
150	100	Lipton	with	0.91	0.91	23.3	0.92	1.99	0.53	0.20	n.d.	3.64	5.15	5.15	9.47	0.74	2.52	0.06	13.4	
150	100	Lipton	with	1.12	1.12	12.8	0.73	1.80	0.66	0.33	0.18	4.73	4.53	4.53	5.70	0.47	2.25	0.09	8.73	
MEAN				0.89	0.88	16.3	0.70	1.61	0.50	0.22	0.12	3.39	4.82	4.82	7.73	0.55	2.78	0.07	11.5	
150	100	Lipton	without	0.88	0.56	18.0	0.64	1.33	0.43	0.20	0.09	2.70	3.27	3.27	8.17	0.54	2.92	0.06	12.0	
150	100	Lipton	with	1.03	0.78	18.8	0.64	1.32	0.41	0.16	n.d.	2.53	3.35	3.35	7.47	0.41	2.70	0.08	10.9	
150	100	Lipton	without	1.03	1.30	11.4	0.62	1.60	0.41	0.21	0.10	2.94	3.77	3.77	7.98	1.14	3.30	0.11	13.1	
150	100	Lipton	with	0.65	0.65	18.1	1.25	3.26	0.63	0.24	0.11	5.64	3.22	3.22	6.49	0.76	2.55	0.05	10.4	
MEAN				0.82	0.82	16.6	0.79	1.88	0.47	0.20	0.10	3.45	3.40	3.40	7.53	0.71	2.87	0.08	11.6	
150	200	Bristol	with	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.32	0.25	0.99	0.08	3.82	
150	200	Bristol	with	0.83	0.83	8.51	0.09	0.66	0.24	0.07	n.d.	1.07	3.90	3.90	5.70	0.59	3.22	0.12	9.85	
150	200	Bristol	with	0.86	0.86	23.3	0.16	0.86	0.33	0.12	n.d.	1.42	5.77	5.77	6.71	0.53	3.23	0.10	10.9	
150	200	Bristol	with	0.36	0.36	7.13	0.11	1.31	0.34	0.07	n.d.	1.91	3.74	3.74	4.82	0.46	2.13	0.09	7.92	
MEAN				0.75	0.68	13.0	0.12	0.95	0.30	0.09	n.d.	1.47	4.47	4.47	4.89	0.46	2.40	0.10	8.13	
150	200	Bristol	with	0.74	0.74	12.1	0.33	1.83	0.51	0.08	n.d.	2.84	3.83	3.83	4.79	0.45	2.18	0.07	7.69	
150	200	Bristol	with	0.76	0.76	19.1	0.32	0.75	0.21	0.12	n.d.	1.61	4.18	4.18	8.48	0.61	2.84	0.07	12.4	
150	200	Bristol	with	0.86	0.86	11.9	0.18	1.17	0.29	0.03	n.d.	1.76	3.56	3.56	4.90	0.43	2.10	0.18	8.00	
150	200	Bristol	with	1.19	1.19	12.0	0.06	0.38	0.15	0.17	n.d.	0.62	6.16	6.16	7.49	0.62	4.34	0.10	13.3	
MEAN				1.03	0.89	13.8	0.22	1.04	0.29	0.10	n.d.	1.71	4.43	4.43	6.41	0.53	2.87	0.11	10.3	

Table A.13: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL
$\mu\text{mol g}^{-1}$																		
2002/2003																		
150	200	<i>Lipton</i>	with	0.86	0.77	8.7	0.32	0.69	0.24	0.09	n.d.	1.39	2.95	8.79	0.55	3.52	0.11	13.1
150	200	<i>Lipton</i>	with		0.68	17.2	0.10	0.84	0.25	0.35	0.13	1.28	4.42	8.22	0.56	2.66	0.10	11.8
150	200	<i>Lipton</i>	with		0.89	10.4	1.21	2.38	0.62	0.06	0.04	4.70	3.33	6.20	0.39	1.94	0.08	8.94
150	200	<i>Lipton</i>	with	—	1.23	16.7	0.35	0.73	0.22	0.06	n.d.	1.40	4.70	5.15	0.40	3.19	0.10	9.41
MEAN					0.89	13.2	0.50	1.16	0.33	0.14	0.08	2.19	3.85	7.09	0.47	2.83	0.10	10.8
150	200	<i>Lipton</i>	nwithout	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	7.89	0.47	2.75	0.09	11.5
150	200	<i>Lipton</i>	nwithout	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	10.5	0.75	3.16	0.15	14.9
150	200	<i>Lipton</i>	nwithout	.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	7.43	0.54	2.22	0.11	11.2
150	200	<i>Lipton</i>	nwithout	—	0.95	8.60	0.41	0.80	0.27	0.11	n.d.	1.59	3.42	10.2	0.55	3.27	0.09	14.7
MEAN					0.95	8.60	0.41	0.80	0.27	0.11	n.a.	1.59	3.42	9.01	0.58	2.85	0.11	13.1

note: n.a. n ot available; m.v. – missing value; n.d. – not detected; cys – cysteine; γ -glu-cys – γ -glutamyl cysteine; GSH – glutathione; Prog – progoitrin; GBN – glucobrassicinapin; GN – gluconapin; GB – gluconapin; GNT – gluconasturtiin; GSL – total glucosinolates; Prot-S – S protein; GNF – gluconapoleiferin; 4-HGB – 4-hydroxy-gluco Brassicic acid

Table A.14: Organic sulphur compounds in leaf and seed samples, Aberdeen, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves								Seeds							
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL	
				$\mu\text{mol g}^{-1}$															
				2000/2001															
0	100	Bristol	with	1.34	0.53	22.7	0.14	0.77	0.11	0.62	0.20	4.88	2.49	2.82	0.39	1.90	0.02	5.97	
0	100	Bristol085	with		0.14	19.6	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.33	0.27	1.77	0.02	5.20	
0	100	Bristol07	with		0.25	22.1	0.08	0.90	0.12	0.26	0.31	4.52	3.09	2.98	0.49	2.12	0.02	6.56	
0	100	Bristol14	with		0.49	8.21	0.44	1.38	0.30	0.37	0.33	4.84	2.93	2.98	0.30	1.89	0.03	6.08	
MEAN				1.10	0.35	18.2	0.22	1.02	0.18	0.42	0.28	4.75	2.79	2.76	0.36	1.92	0.02	5.95	
0	100	Bristol	nwithout	a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.08	0.50	2.00	0.02	6.80	
0	100	Bristol	wf0out		0.14	23.9	0.60	1.72	0.33	1.31	0.40	2.05	n.a.	2.48	0.06	1.82	0.02	5.52	
0	100	Bristol	wf6out		0.26	23.2	0.45	1.72	0.22	1.08	0.22	2.13	1.68	2.70	0.41	1.81	0.03	5.96	
0	100	Bristol	wf8out		0.44	12.5	0.33	2.52	0.34	0.64	0.40	3.26	2.66	2.70	0.45	2.11	0.02	6.83	
MEAN				0.79	0.28	19.8	0.46	1.99	0.30	1.01	0.34	2.48	2.17	2.88	0.36	1.93	0.02	6.28	
0	100	Lipton08	with		0.31	12.6	0.80	1.60	0.59	0.47	0.28	3.53	2.07	2.94	0.35	1.51	0.02	5.92	
0	100	Lipton32	with		0.11	22.2	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.44	0.20	1.30	0.02	4.80	
0	100	Lipton22	with		0.24	27.0	0.36	0.87	0.27	0.27	0.26	5.24	1.82	2.91	0.33	1.52	0.03	5.70	
0	100	Lipton	with	a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	2.70	2.91	0.33	1.52	0.03	5.70	
MEAN				1.37	0.22	20.6	0.58	1.24	0.43	0.37	0.27	4.38	1.95	2.75	0.28	1.41	0.03	5.35	
0	100	Lipton	nwithout	a.	n.a.	n.a.	0.46	1.47	0.38	0.69	0.25	2.67	n.a.	2.83	0.43	1.47	0.03	5.76	
0	100	Lipton	wf4out		0.26	22.7	0.60	2.81	0.82	0.33	0.18	4.07	0.53	3.27	0.41	1.64	0.03	6.47	
0	100	Lipton	nwithout	a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.21	0.40	1.61	0.03	6.17	
0	100	Lipton	wf8out		0.48	14.5	0.40	0.94	0.26	0.52	0.33	2.34	2.23	3.21	0.38	1.56	0.01	6.12	
MEAN				0.78	0.37	18.6	0.49	1.74	0.49	0.51	0.25	3.03	1.39	3.16	0.41	1.57	0.03	6.13	
0	200	Bristol08	with		0.41	20.4	0.18	0.78	0.08	0.99	0.17	4.38	2.36	2.01	0.26	1.17	0.03	4.54	
0	200	Bristol11	with		0.23	15.4	0.68	1.48	0.28	0.77	0.24	5.57	0.80	1.65	0.14	1.07	0.03	3.66	
0	200	Bristol38	with		0.25	31.5	0.41	1.47	0.22	0.62	0.24	3.71	1.75	2.00	0.29	1.34	0.03	4.56	
0	200	Bristol86	with		0.60	11.1	0.32	1.13	0.22	0.48	0.26	3.79	1.87	2.00	0.25	1.64	0.02	5.29	
MEAN				1.11	0.37	19.6	0.40	1.21	0.20	0.71	0.23	4.36	1.68	2.05	0.23	1.31	0.03	4.51	
0	200	Bristol	wf4out		0.26	24.6	0.59	1.98	0.32	0.67	0.26	2.51	1.98	2.47	0.37	1.52	0.03	5.41	
0	200	Bristol	wf6out		0.28	24.4	0.54	1.61	0.25	2.31	0.37	4.11	5.35	1.95	0.29	1.23	0.03	4.22	
0	200	Bristol	wf8out		0.32	21.9	0.32	1.45	0.20	0.77	0.29	3.73	5.95	2.31	0.32	1.29	0.03	4.86	
0	200	Bristol	wf8out		0.69	12.2	0.46	1.34	0.21	1.04	0.26	2.85	2.23	2.31	0.35	1.74	n.d.	5.63	
MEAN				1.11	0.39	20.8	0.48	1.60	0.24	1.20	0.30	3.30	3.89	2.36	0.33	1.44	0.03	5.03	

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds						
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL		
				μmol g ⁻¹																
				2000/2001																
0	200	Lipton	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	0.35	1.53	0.02	6.06
0	200	Lipto22	with	0.50	12.7	0.81	n.a.	0.29	0.33	0.29	0.20	3.85	1.57	2.62	0.21	1.26	0.03	4.96		
0	200	Lipto00	with	0.67	9.47	0.41	0.78	0.34	0.21	0.34	0.20	4.86	1.96	3.53	0.34	1.55	0.03	6.29		
0	200	Lipto01	with	<u>0.30</u>	<u>26.0</u>	<u>0.47</u>	<u>1.50</u>	<u>0.54</u>	<u>0.42</u>	<u>0.41</u>	<u>5.52</u>	<u>3.20</u>	—	<u>0.33</u>	<u>1.93</u>	<u>0.02</u>	<u>6.47</u>			
MEAN				1.08	16.1	0.56	1.07	0.39	0.32	0.27	4.74	1.77	3.11	0.31	1.57	0.02	5.94			
0	200	Lipton	n without	n.a.	n.a.	0.69	1.48	0.74	0.46	0.74	0.20	4.69	n.a.	3.50	0.43	1.64	0.02	6.73		
0	200	Lipton	with	0.24	27.2	0.87	1.79	0.79	0.42	0.79	0.31	3.10	1.02	2.81	0.31	1.44	0.03	5.54		
0	200	Lipton	with	0.24	22.2	1.20	1.96	0.78	0.56	0.78	0.28	2.51	1.60	4.42	0.56	1.87	0.03	8.17		
0	200	Lipton	without	<u>0.45</u>	<u>16.1</u>	<u>1.03</u>	<u>1.88</u>	<u>0.63</u>	<u>0.43</u>	<u>0.27</u>	<u>3.62</u>	<u>3.67</u>	—	<u>0.36</u>	<u>1.62</u>	<u>0.04</u>	<u>6.61</u>			
MEAN				0.80	21.8	0.95	1.78	0.73	0.47	0.27	3.48	1.42	3.60	0.42	1.64	0.03	6.76			
100	100	Bristol	with	0.46	20.5	0.25	1.68	0.69	0.26	0.69	0.33	7.79	0.75	3.83	0.65	2.37	0.02	7.74		
100	100	Bristol	with	0.20	27.4	0.50	0.97	1.03	0.21	1.03	0.21	3.19	1.93	3.10	0.38	2.26	0.02	6.72		
100	100	Bristol	with	0.23	20.9	0.46	1.53	0.51	0.33	0.51	0.23	4.53	2.45	3.58	0.56	2.56	0.03	7.90		
100	100	Bristol	with	<u>n.a.</u>	<u>n.a.</u>	<u>0.57</u>	<u>1.68</u>	<u>0.60</u>	<u>0.42</u>	<u>0.29</u>	<u>3.75</u>	<u>3.59</u>	—	<u>0.46</u>	<u>2.52</u>	<u>0.02</u>	<u>7.73</u>			
MEAN				1.32	22.9	0.45	1.47	0.71	0.30	0.27	4.82	1.71	3.53	0.51	2.43	0.02	7.52			
100	100	Bristol	without	0.21	23.6	0.99	2.78	2.20	0.52	2.20	0.52	3.58	2.55	3.66	0.72	2.47	0.01	8.12		
100	100	Bristol	n without	n.a.	n.a.	0.31	1.40	0.66	0.27	0.66	0.28	3.45	n.a.	3.39	0.59	2.32	0.03	7.55		
100	100	Bristol	with	0.29	19.1	0.44	1.73	1.15	0.27	1.15	0.23	3.82	n.a.	3.74	0.79	2.46	0.02	8.32		
100	100	Bristol	without	<u>0.44</u>	<u>8.70</u>	<u>0.41</u>	<u>1.45</u>	<u>0.98</u>	<u>0.26</u>	<u>0.26</u>	<u>3.97</u>	<u>3.80</u>	—	<u>0.65</u>	<u>2.35</u>	<u>0.02</u>	<u>8.10</u>			
MEAN				1.10	17.1	0.54	1.84	1.25	0.33	0.32	3.70	1.71	3.65	0.69	2.40	0.02	8.02			
100	100	Lipto69	with	0.44	11.0	0.66	1.51	0.64	0.56	0.64	0.32	2.93	3.66	3.92	0.49	1.91	0.02	7.45		
100	100	Lipto50	with	0.12	20.2	0.97	1.20	0.54	0.54	0.28	4.51	1.68	2.99	0.33	1.50	0.02	5.76			
100	100	Lipto13	with	0.20	21.8	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.73	0.51	1.83	0.04	7.17		
100	100	Lipto08	with	<u>0.50</u>	<u>19.7</u>	<u>0.76</u>	<u>1.14</u>	<u>0.53</u>	<u>0.53</u>	<u>0.41</u>	<u>0.29</u>	<u>4.00</u>	<u>3.38</u>	—	<u>0.36</u>	<u>1.80</u>	<u>0.02</u>	<u>6.48</u>		
MEAN				1.35	18.2	0.79	1.28	0.44	0.54	0.28	3.81	2.67	3.50	0.42	1.76	0.03	6.72			
100	100	Lipton	without	0.26	22.8	0.52	1.14	0.37	0.42	0.37	0.19	3.68	1.32	3.60	0.59	1.74	0.02	7.16		
100	100	Lipton	with	0.21	32.7	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.38	0.86	1.71	0.03	7.01		
100	100	Lipton	n without	n.a.	n.a.	0.53	2.02	0.63	0.63	0.39	0.45	3.58	n.a.	4.33	0.64	1.99	0.03	8.15		
100	100	Lipton	without	<u>0.28</u>	<u>17.5</u>	<u>0.53</u>	<u>2.02</u>	<u>0.39</u>	<u>0.63</u>	<u>0.45</u>	<u>3.58</u>	<u>3.94</u>	—	<u>0.49</u>	<u>1.66</u>	<u>0.01</u>	<u>6.97</u>			
MEAN				1.16	24.3	0.53	1.58	0.38	0.52	0.32	3.63	2.43	3.81	0.64	1.77	0.02	7.32			

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds							
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL
				$\mu\text{mol g}^{-1}$														
				2000/2001														
100	200	<i>Bristol</i>	with	1.42	0.54	21.5	0.38	1.42	0.19	0.86	0.24	3.81	5.01	4.02	0.61	2.36	0.01	8.32
100	200	<i>Bristol</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	m.a.	.v.	m.v.	m.v.	m.v.	m.v.
100	200	<i>Bristol</i>	with	1.15	0.27	27.1	0.30	0.83	0.13	0.68	0.37	6.08	2.45	4.42	0.70	2.78	0.03	9.00
100	200	<i>Bristol</i>	with	—	0.69	6.68	0.54	0.89	0.24	0.45	0.26	7.40	14.37	—	0.52	2.71	0.01	8.80
MEAN				1.27	0.50	18.5	0.41	1.05	0.19	0.66	0.29	5.76	2.95	4.24	0.61	2.62	0.02	8.70
100	200	<i>Bristol</i>	without	0.39	0.21	11.2	0.63	1.46	0.29	0.68	0.26	4.29	0.35	4.17	0.79	2.41	0.02	8.87
100	200	<i>Bristol</i>	without	0.21	0.24	24.2	0.63	1.31	0.18	3.17	0.26	3.56	n.a.	3.82	0.54	2.40	0.02	8.02
100	200	<i>Bristol</i>	without	0.24	0.24	25.1	1.13	2.28	0.42	2.52	0.28	2.85	1.70	4.04	0.70	2.36	0.03	8.51
100	200	<i>Bristol</i>	without	0.42	0.42	19.0	0.58	1.78	0.32	0.78	0.25	2.76	4.00	—	0.79	2.41	0.02	9.03
MEAN				1.13	0.31	19.9	0.74	1.71	0.30	1.79	0.26	3.37	2.04	4.13	0.70	2.39	0.02	8.61
100	200	<i>Lipton</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.92	0.46	1.69	0.02	7.22
100	200	<i>Lipton</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.40	0.53	1.70	0.04	6.62
100	200	<i>Lipton</i>	with	0.24	0.24	16.9	0.50	1.03	0.36	0.34	0.33	2.88	0.56	4.44	0.48	2.07	0.02	8.05
100	200	<i>Lipton</i>	with	0.74	0.74	7.10	0.72	0.84	0.32	0.16	0.27	2.59	3.36	—	0.33	1.54	0.02	6.18
MEAN				1.17	0.49	12.0	0.61	0.94	0.34	0.25	0.30	2.74	1.96	3.74	0.45	1.75	0.02	7.02
100	200	<i>Lipton</i>	without	0.31	0.31	14.3	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.95	0.54	2.00	0.02	7.62
100	200	<i>Lipton</i>	without	0.28	0.28	25.0	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	4.63	0.56	2.21	0.03	8.76
100	200	<i>Lipton</i>	without	0.27	0.27	21.5	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.94	0.53	1.87	0.03	7.56
100	200	<i>Lipton</i>	without	m.v.	0.43	14.8	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	4.83	—	0.55	2.15	0.02	8.64
MEAN				1.29	0.32	18.9	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	4.34	0.55	2.06	0.03	8.14
				2001/2002														
0	100	<i>Bristol</i>	with	0.71	0.11	17.8	0.10	2.04	0.32	0.07	0.25	2.83	2.98	2.46	0.44	1.40	n.d.	4.43
0	100	<i>Bristol</i>	with	0.77	0.12	20.6	0.07	1.35	0.26	0.06	0.17	1.99	2.55	2.68	0.47	1.69	0.01	5.49
0	100	<i>Bristol</i>	with	0.66	0.14	17.5	0.07	1.31	0.30	0.09	0.18	1.99	2.71	2.65	0.31	1.51	0.05	5.17
0	100	<i>Bristol</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	3.83	—	0.63	1.78	0.05	6.63
MEAN				0.71	0.12	18.6	0.08	1.57	0.29	0.07	0.20	2.27	2.75	2.79	0.46	1.60	0.04	5.43

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				Leaves				Seeds						
				γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL	
2001/2002																		
μmol g ⁻¹																		
0	100	Bristol	without	0.89	0.15	24.1	0.70	4.17	0.87	0.17	0.38	6.37	2.60	2.90	0.70	1.51	0.02	5.48
0	100	Bristol	without	0.52	0.31	16.8	0.49	2.69	0.60	0.22	0.23	4.23	2.41	5.04	1.05	2.70	0.05	10.1
0	100	Bristol	without	0.66	0.11	19.4	0.16	2.08	0.56	0.13	0.21	3.19	3.85	3.94	0.75	2.44	0.04	8.15
0	100	Bristol	with	—	<u>0.10</u>	<u>19.9</u>	<u>1.10</u>	<u>7.32</u>	<u>1.44</u>	<u>0.44</u>	<u>0.68</u>	<u>11.2</u>	<u>2.75</u>	—	<u>0.64</u>	<u>1.55</u>	<u>0.04</u>	<u>6.09</u>
MEAN				0.64	0.17	20.0	0.61	4.06	0.87	0.24	0.38	6.26	2.91	3.78	0.78	2.05	0.04	7.46
0	100	Lipton	with	0.85	0.15	20.8	0.26	0.86	0.24	0.08	0.18	1.72	2.61	2.42	0.45	0.99	0.02	3.88
0	100	Lipton	with	0.55	0.14	23.3	0.48	1.63	0.33	0.09	0.26	2.92	2.47	3.02	0.50	1.23	0.05	5.42
0	100	Lipton	with	0.55	0.10	20.1	0.62	1.67	0.26	0.09	0.23	3.05	2.68	2.95	0.40	1.39	0.06	5.08
0	100	Lipton	with	—	<u>0.11</u>	<u>17.9</u>	<u>1.09</u>	<u>3.58</u>	<u>0.69</u>	<u>0.20</u>	<u>0.61</u>	<u>6.71</u>	<u>3.90</u>	—	<u>0.54</u>	<u>1.42</u>	<u>0.08</u>	<u>5.84</u>
MEAN				0.56	0.12	20.5	0.61	1.94	0.38	0.12	0.32	3.60	2.92	2.89	0.47	1.26	0.05	5.06
0	100	Lipton	without	0.70	0.11	19.6	1.55	4.85	1.08	0.26	0.38	8.25	2.64	3.70	0.86	1.30	0.04	6.23
0	100	Lipton	without	0.50	0.08	20.0	0.57	2.22	0.44	0.09	0.28	3.75	2.94	3.15	0.58	1.26	0.02	5.51
0	100	Lipton	without	0.58	0.10	18.9	0.69	2.18	0.34	0.17	0.22	3.75	2.98	4.04	0.69	1.77	0.09	7.14
0	100	Lipton	with	—	<u>0.13</u>	<u>20.4</u>	<u>0.63</u>	<u>1.83</u>	<u>0.29</u>	<u>0.12</u>	<u>0.18</u>	<u>3.22</u>	<u>2.82</u>	—	<u>0.66</u>	<u>1.57</u>	<u>0.09</u>	<u>6.89</u>
MEAN				0.61	0.10	19.7	0.86	2.77	0.54	0.16	0.26	4.74	2.84	3.68	0.70	1.48	0.06	6.44
0	200	Bristol	with	0.60	0.16	19.2	0.04	0.86	0.21	0.03	0.12	1.31	2.82	2.22	0.39	1.32	0.01	4.15
0	200	Bristol	with	0.78	0.11	21.7	0.16	1.92	0.41	0.10	0.20	2.86	2.36	1.98	0.31	1.08	0.02	3.77
0	200	Bristol	with	0.58	0.13	18.4	0.08	1.05	0.27	0.06	0.14	1.66	2.97	2.15	0.28	1.13	0.03	3.98
0	200	Bristol	with	—	<u>0.12</u>	<u>23.4</u>	<u>0.63</u>	<u>3.31</u>	<u>0.55</u>	<u>0.17</u>	<u>0.47</u>	<u>5.37</u>	<u>2.52</u>	—	<u>0.49</u>	<u>1.79</u>	<u>0.07</u>	<u>6.34</u>
MEAN				0.70	0.13	20.7	0.23	1.79	0.36	0.09	0.23	2.80	2.68	2.47	0.37	1.33	0.03	4.56
0	200	Bristol	without	0.64	0.16	25.8	0.34	4.15	0.69	0.26	0.35	5.78	2.11	2.75	0.56	1.39	0.03	5.17
0	200	Bristol	without	0.53	0.30	20.3	0.26	2.82	0.54	0.25	0.27	4.20	3.22	4.51	1.11	2.45	0.02	9.22
0	200	Bristol	without	0.59	0.14	19.9	0.25	1.83	0.36	0.15	0.19	2.88	1.51	3.56	0.46	1.68	0.07	6.31
0	200	Bristol	with	—	<u>0.16</u>	<u>19.2</u>	<u>0.17</u>	<u>1.77</u>	<u>0.42</u>	<u>0.10</u>	<u>0.15</u>	<u>2.73</u>	<u>2.45</u>	—	<u>0.52</u>	<u>1.44</u>	<u>0.05</u>	<u>5.96</u>
MEAN				0.57	0.19	21.3	0.25	2.64	0.50	0.19	0.24	3.90	2.33	3.52	0.66	1.74	0.04	6.67
0	200	Lipton	with	0.70	0.13	21.7	0.54	1.74	0.36	0.09	0.30	3.19	0.78	1.93	0.26	0.80	0.02	3.00
0	200	Lipton	with	0.78	0.19	24.9	0.26	0.93	0.24	0.10	0.21	1.94	2.90	3.39	0.41	1.33	0.03	5.71
0	200	Lipton	with	0.50	0.15	23.6	0.44	1.36	0.24	0.09	0.21	2.45	2.45	3.71	0.40	1.48	0.09	6.22
0	200	Lipton	with	—	<u>0.16</u>	<u>24.4</u>	<u>1.03</u>	<u>3.15</u>	<u>0.56</u>	<u>0.26</u>	<u>0.62</u>	<u>6.33</u>	<u>3.02</u>	—	<u>0.55</u>	<u>1.50</u>	<u>0.08</u>	<u>6.40</u>
MEAN				0.62	0.16	23.6	0.56	1.80	0.35	0.14	0.33	3.48	2.29	3.20	0.41	1.27	0.06	5.33

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL
				μmol g ⁻¹														
2001/2002																		
0	200	Lipton	without	0.67	0.17	25.3	0.77	2.17	0.62	0.19	0.27	4.16	2.84	3.53	0.65	1.36	0.05	5.84
0	200	Lipton	without	0.58	0.29	24.1	0.58	2.15	0.50	0.15	0.24	3.85	2.79	4.87	0.86	1.93	0.06	8.59
0	200	Lipton	without	0.55	0.17	22.3	0.45	1.61	0.39	0.15	0.21	2.92	2.31	3.59	0.49	1.35	0.05	6.13
0	200	Lipton	with	—	0.16	24.3	0.74	1.71	0.40	0.15	0.23	3.46	2.68	—	0.50	1.56	0.11	6.83
MEAN				0.57	0.20	24.0	0.63	1.91	0.48	0.16	0.24	3.60	2.64	4.02	0.63	1.55	0.07	6.85
100	100	Bristol	with	1.06	0.43	26.8	0.18	1.78	0.37	0.09	0.32	2.82	5.78	3.96	1.00	2.36	0.02	7.55
100	100	Bristol	with	0.99	0.53	30.4	0.19	1.91	0.37	0.09	0.29	2.91	5.60	3.99	0.84	2.15	0.05	7.91
100	100	Bristol	with	1.13	0.33	30.1	0.30	2.45	0.41	0.12	0.30	3.79	8.25	4.38	0.83	2.36	0.08	8.53
100	100	Bristol	with	—	0.22	21.2	0.39	3.87	0.59	0.25	0.55	5.89	3.88	—	1.09	2.47	0.06	9.18
MEAN				0.97	0.38	27.1	0.26	2.50	0.44	0.14	0.37	3.85	6.38	4.20	0.94	2.33	0.05	8.29
100	100	Bristol	without	1.23	0.28	27.9	0.54	4.99	1.12	0.36	0.60	7.73	6.54	5.18	1.47	2.69	0.03	9.79
100	100	Bristol	without	0.89	0.25	23.6	1.26	3.37	0.59	0.16	0.33	5.84	5.32	4.10	0.94	1.64	0.03	7.46
100	100	Bristol	without	0.80	0.21	23.4	0.17	2.43	0.39	0.16	0.36	3.76	4.75	4.82	1.07	2.70	0.03	9.85
100	100	Bristol	with	—	0.22	22.4	0.77	5.62	1.06	0.52	0.60	8.90	5.78	—	1.17	2.46	0.08	9.91
MEAN				0.90	0.24	24.3	0.68	4.10	0.79	0.30	0.47	6.56	5.59	4.79	1.16	2.37	0.04	9.25
100	100	Lipton	with	1.06	0.34	26.3	0.84	2.15	0.40	0.10	0.34	3.99	3.25	3.67	0.86	1.48	0.04	6.21
100	100	Lipton	with	1.12	0.45	27.8	0.77	2.46	0.40	0.11	0.30	4.21	4.88	3.94	0.82	1.64	0.03	6.64
100	100	Lipton	with	0.95	0.43	29.8	0.72	2.28	0.36	0.10	0.40	3.95	4.60	3.87	0.60	1.68	0.07	6.55
100	100	Lipton	with	—	0.37	29.1	m.v.	m.v.	m.v.	m.v.	m.v.	m.v.	4.14	—	0.86	1.66	0.07	7.38
MEAN				0.99	0.40	28.2	0.78	2.30	0.39	0.11	0.35	4.05	4.24	3.90	0.79	1.62	0.05	6.69
100	100	Lipton	without	1.00	0.25	28.4	1.74	6.14	1.13	0.33	0.58	10.2	3.91	4.31	1.29	1.79	0.07	8.11
100	100	Lipton	without	0.58	0.10	24.1	1.03	2.87	0.56	0.17	0.31	5.06	2.75	3.80	0.56	1.40	0.05	6.44
100	100	Lipton	without	0.71	0.21	23.7	0.83	2.62	0.34	0.19	0.39	4.43	5.04	4.03	0.89	1.82	0.04	7.40
100	100	Lipton	with	—	0.22	21.4	1.42	3.21	0.52	0.19	0.28	5.82	5.48	—	1.04	2.21	0.08	9.47
MEAN				0.72	0.20	24.4	1.26	3.71	0.64	0.22	0.39	6.38	4.22	4.38	0.94	1.80	0.06	7.86
100	200	Bristol	with	1.19	0.38	31.0	0.21	1.45	0.30	0.09	0.23	2.41	6.11	4.54	0.85	2.43	0.04	8.24
100	200	Bristol	with	1.18	0.33	29.9	0.12	1.65	0.30	0.10	0.17	2.44	3.40	4.94	0.77	2.90	0.03	8.99
100	200	Bristol	with	0.76	0.29	24.7	0.24	1.37	0.06	0.13	0.21	2.07	4.90	4.96	0.78	2.68	0.08	9.29
100	200	Bristol	with	—	0.31	31.9	m.v.	m.v.	m.v.	m.v.	m.v.	m.v.	4.18	—	0.85	2.86	0.11	9.37
MEAN				1.03	0.33	29.3	0.19	1.49	0.22	0.11	0.20	2.31	4.80	4.80	0.81	2.72	0.06	8.97

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds						
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL		
				$\mu\text{mol g}^{-1}$																
2001/2002																				
100	200	Bristol	w0182ut	0.28	28.5	1.06	4.39	1.12	0.26	0.43	7.34	5.08	4.89	1.21	2.35	0.09	9.27			
100	200	Bristol	w0182ut	0.12	28.7	0.17	1.24	0.30	0.12	0.16	1.99	6.71	2.39	0.42	1.12	0.02	4.56			
100	200	Bristol	w0184ut	0.20	24.9	0.45	3.01	0.65	0.29	0.28	4.77	4.82	5.04	0.99	2.82	0.07	9.81			
100	200	Bristol	w0179ut	0.24	26.7	0.46	2.49	0.55	0.25	0.21	4.12	6.26	—	1.06	2.20	0.08	9.15			
MEAN				0.86	27.2	0.53	2.78	0.66	0.23	0.27	4.55	5.71	4.24	0.92	2.12	0.06	8.20			
100	200	Lipton 1.29 with		0.38	32.2	0.49	1.79	0.32	0.11	0.23	3.19	5.36	4.31	0.65	1.65	0.07	6.96			
100	200	Lipton 0.62 with		0.60	20.9	0.22	1.43	0.33	0.11	0.17	2.26	5.49	4.68	0.78	2.03	0.06	8.48			
100	200	Lipton 0.98 with		0.30	33.8	0.49	1.64	0.09	0.10	0.28	2.87	6.52	5.48	0.84	2.30	0.11	9.29			
100	200	Lipton 0.82 with		0.45	29.7	m.v.	m.v.	m.v.	m.v.	m.v.	m.v.	4.36	—	0.73	1.64	0.06	7.43			
MEAN				0.93	29.1	0.40	1.62	0.25	0.11	0.23	2.77	5.79	4.71	0.75	1.91	0.08	8.04			
100	200	Lipton	w106ut	0.27	30.5	0.21	2.55	0.59	0.20	0.36	3.91	4.51	4.72	0.98	1.68	0.06	7.78			
100	200	Lipton	w105ut	0.07	32.1	0.08	2.30	0.57	0.13	0.22	3.30	3.88	3.42	0.65	1.85	0.02	6.77			
100	200	Lipton	w0170ut	0.24	27.8	1.23	3.19	0.42	0.18	0.44	5.54	6.21	5.09	1.05	1.99	0.06	8.91			
100	200	Lipton	w0179ut	0.36	30.8	0.87	2.49	0.35	0.21	0.28	4.41	7.22	—	0.88	2.40	0.11	10.1			
MEAN				0.90	30.3	0.60	2.63	0.48	0.18	0.33	4.29	5.45	4.79	0.89	1.98	0.06	8.39			
2002/2003																				
				Leaves										Seeds						
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	GB	GSL		
				$\mu\text{mol g}^{-1}$																
0	100	Bristol/0.80 with		1.23	20.0	0.34	1.94	0.36	0.11	n.d.	3.06	2.31	3.88	0.78	3.15	1.13	9.65			
0	100	Bristol/0.85 with		1.18	10.5	0.29	2.62	0.55	0.09	0.12	4.11	2.65	3.38	0.56	1.95	1.77	7.96			
0	100	Bristol/0.91 with		1.07	11.2	1.40	3.41	0.64	0.10	0.10	5.95	1.89	4.09	0.70	2.45	1.61	9.34			
0	100	Bristol/1.01 with		1.09	11.0	0.29	2.77	0.63	0.14	0.23	4.57	14.26	—	0.70	2.76	1.91	9.73			
MEAN				0.89	13.2	0.58	2.68	0.54	0.11	0.15	4.42	2.14	3.90	0.68	2.58	1.60	9.17			
0	100	Bristol	w0182ut	1.14	10.2	0.51	3.39	0.63	0.24	0.16	5.51	5.02	4.21	0.76	2.28	2.01	9.69			
0	100	Bristol	w0195ut	1.26	9.23	0.23	2.37	0.41	0.14	0.20	3.83	2.63	4.62	0.95	3.16	1.96	11.2			
0	100	Bristol	w0195ut	1.10	19.0	1.27	2.97	0.60	0.17	0.30	5.53	1.92	6.97	0.81	2.55	1.90	12.6			
0	100	Bristol	w0182ut	0.97	8.45	0.27	2.76	0.65	0.14	0.18	4.47	24.56	—	0.66	2.59	1.87	10.2			
MEAN				0.90	11.7	0.57	2.87	0.57	0.17	0.21	4.83	3.02	5.09	0.79	2.64	1.94	10.9			

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds			
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	P rog	GBN	GN	4-HGB
2002/2003																	
0	100	Lipton	with	0.68	1.04	18.5	0.32	1.97	0.49	0.12	0.17	3.37	1.95	0.76	1.98	1.21	8.76
0	100	Lipton	with	0.60	0.95	18.2	0.25	1.91	0.50	0.14	0.20	3.35	2.27	0.38	1.52	2.13	7.56
0	100	Lipton	with	0.77	1.06	9.21	0.91	3.20	1.07	0.17	0.26	6.25	2.56	0.65	1.81	1.70	8.24
0	100	Lipton0.80	with	—	<u>1.06</u>	<u>9.72</u>	<u>m.v.</u>	<u>m.v.</u>	<u>m.v.</u>	<u>m.v.</u>	<u>m.v.</u>	<u>m.v.</u>	<u>3.88</u>	<u>0.54</u>	<u>1.57</u>	<u>1.96</u>	<u>8.19</u>
MEAN				0.71	1.03	13.9	0.49	2.36	0.69	0.14	0.21	4.32	2.26	0.58	1.72	1.75	8.19
0	100	Lipton	without	0.75	0.97	10.5	0.40	2.30	0.82	0.05	n.d.	4.86	4.20	0.85	1.86	2.41	9.73
0	100	Lipton	without	0.70	1.31	10.0	0.34	3.66	1.10	0.22	0.24	6.05	2.59	0.50	1.54	0.57	6.34
0	100	Lipton	without	0.77	1.19	8.43	0.36	2.33	0.57	0.13	0.10	3.86	2.38	0.70	1.42	2.10	8.36
0	100	Lipton	without	—	<u>1.00</u>	<u>11.2</u>	<u>0.33</u>	<u>2.91</u>	<u>0.78</u>	<u>0.12</u>	<u>0.12</u>	<u>4.68</u>	<u>4.95</u>	<u>0.91</u>	<u>2.94</u>	<u>1.93</u>	<u>13.2</u>
MEAN				0.77	1.12	10.0	0.36	2.80	0.82	0.13	0.15	4.86	3.40	0.74	1.94	1.75	9.40
0	200	Bristol0.55	with	0.68	1.17	10.1	0.45	2.14	0.42	0.10	n.d.	3.47	2.68	0.55	2.23	1.15	7.44
0	200	Bristol	with	0.73	1.33	11.5	0.26	1.64	0.28	0.15	0.14	2.82	2.66	0.32	1.54	2.37	7.12
0	200	Bristol	with	0.73	1.35	9.34	1.91	1.55	0.27	0.11	0.13	4.20	2.81	0.58	1.62	1.99	7.99
0	200	Bristol0.62	with	—	<u>1.30</u>	<u>8.42</u>	<u>0.28</u>	<u>2.10</u>	<u>0.44</u>	<u>0.10</u>	<u>0.12</u>	<u>3.41</u>	<u>3.15</u>	<u>0.46</u>	<u>1.90</u>	<u>2.00</u>	<u>7.97</u>
MEAN				0.64	1.29	9.83	0.73	1.86	0.35	0.12	0.13	3.47	2.82	0.48	1.82	1.87	7.63
0	200	Bristol	without	0.71	1.48	12.6	0.50	2.15	0.42	0.12	n.d.	3.57	2.72	0.62	2.11	1.21	7.86
0	200	Bristol	without	0.73	1.39	9.28	0.37	2.49	0.46	0.14	0.12	3.98	2.59	0.50	1.57	1.38	7.08
0	200	Bristol	without	—	1.24	7.83	0.50	3.23	0.75	0.08	0.08	5.35	2.77	0.84	1.60	1.86	7.46
0	200	Bristol	without	—	<u>1.16</u>	<u>11.9</u>	<u>0.49</u>	<u>3.93</u>	<u>0.93</u>	<u>0.17</u>	<u>0.18</u>	<u>6.39</u>	<u>3.13</u>	<u>0.51</u>	<u>2.00</u>	<u>1.26</u>	<u>8.25</u>
MEAN				0.67	1.32	10.4	0.47	2.95	0.64	0.13	0.13	4.82	2.80	0.62	1.82	1.43	7.66
0	200	Lipton0.50	with	0.51	1.16	8.63	0.34	1.86	0.52	0.09	n.d.	3.07	2.77	0.51	1.55	1.62	7.71
0	200	Lipton	with	0.59	1.40	8.55	0.27	2.02	0.57	0.11	0.14	3.45	2.61	0.41	1.70	2.77	8.87
0	200	Lipton0.65	with	—	1.24	8.09	0.45	2.24	0.55	0.12	n.d.	3.71	3.11	0.37	1.22	2.19	7.56
0	200	Lipton0.70	with	—	<u>1.52</u>	<u>12.6</u>	<u>0.33</u>	<u>2.51</u>	<u>0.72</u>	<u>0.15</u>	<u>0.16</u>	<u>4.29</u>	<u>4.83</u>	<u>0.63</u>	<u>2.34</u>	<u>1.85</u>	<u>9.29</u>
MEAN				0.59	1.33	9.46	0.35	2.16	0.59	0.12	0.15	3.63	3.33	0.48	1.70	2.11	8.36
0	200	Lipton	without	0.60	1.49	9.83	0.55	2.37	0.65	0.20	0.18	4.34	2.54	0.59	1.34	2.46	8.66
0	200	Lipton	without	0.49	1.14	18.8	0.30	2.29	0.57	0.09	0.00	3.58	2.55	0.47	1.82	1.74	9.78
0	200	Lipton	without	0.61	1.19	9.39	0.33	2.42	0.64	0.20	0.15	4.11	2.17	0.49	1.29	2.01	7.94
0	200	Lipton	without	—	<u>1.52</u>	<u>12.1</u>	<u>0.40</u>	<u>2.01</u>	<u>0.63</u>	<u>0.21</u>	<u>0.24</u>	<u>3.80</u>	<u>3.74</u>	<u>0.32</u>	<u>1.77</u>	<u>2.14</u>	<u>8.84</u>
MEAN				0.57	1.33	12.5	0.40	2.27	0.62	0.17	0.14	3.96	2.50	0.47	1.55	2.09	8.80

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ -glu-cys	GSH	P	rog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB
2002/2003																		
				$\mu\text{mol g}^{-1}$														
100	100	Bristol	with	0.95	1.06	10.6	0.33	2.37	0.49	0.09	0.17	3.76	2.45	5.66	0.86	3.30	1.46	11.8
100	100	Bristol	with	1.45	1.01	8.60	0.35	2.49	0.52	0.10	0.11	4.15	2.15	4.58	0.70	2.69	1.93	10.4
100	100	Bristol	with	1.00	0.94	19.6	1.22	3.92	0.84	0.14	0.14	6.85	0.42	7.75	1.03	3.06	2.27	14.5
100	100	Bristol0.95	with	—	0.95	10.4	0.37	2.93	0.70	0.16	0.15	4.69	2.66	—	0.70	3.00	1.28	9.36
MEAN				1.09	0.99	12.3	0.57	2.93	0.64	0.12	0.14	4.86	1.91	5.56	0.82	3.01	1.73	11.5
100	100	Bristol	without	1.25	0.88	9.82	0.42	2.92	0.58	0.16	0.14	4.68	6.37	6.66	1.04	2.87	2.36	13.5
100	100	Bristol	without	0.98	1.16	10.5	0.29	2.63	0.49	n.d.	0.13	4.08	2.92	5.47	0.86	3.27	2.05	12.2
100	100	Bristol	without	0.96	0.91	8.51	0.30	3.91	0.83	0.26	0.16	5.55	2.12	4.39	0.85	2.80	1.89	10.5
100	100	Bristol	without	—	0.87	9.65	0.33	3.71	0.80	0.18	0.17	5.99	2.65	—	0.63	2.58	1.41	10.1
MEAN				1.05	0.96	9.62	0.34	3.29	0.67	0.20	0.15	5.08	3.53	5.39	0.84	2.88	1.93	11.6
100	100	Lipton	with	0.74	1.13	9.55	0.38	2.74	0.80	0.17	0.15	4.61	2.13	4.29	0.84	2.02	1.64	9.28
100	100	Lipton	with	0.77	1.04	11.2	1.10	3.22	0.85	0.15	0.21	6.10	1.56	4.56	0.53	2.09	1.15	8.57
100	100	Lipton	with	0.83	1.10	10.8	1.31	1.64	0.49	0.06	0.08	3.78	2.10	4.24	0.82	1.82	2.05	9.61
100	100	Lipton0.83	with	—	0.90	9.73	0.30	2.07	0.60	0.10	n.d.	3.43	2.47	—	0.82	2.02	2.12	9.59
MEAN				0.79	1.04	10.3	0.77	2.42	0.69	0.12	0.15	4.48	2.06	4.37	0.75	1.99	1.74	9.26
100	100	Lipton	without	0.82	1.03	8.82	0.47	3.16	0.82	0.26	0.20	5.44	1.13	4.88	0.85	1.78	2.74	10.7
100	100	Lipton	without	—	0.93	9.20	0.22	3.21	0.83	0.13	n.d.	4.81	2.81	6.07	0.78	2.50	1.73	11.5
100	100	Lipton	without	0.82	0.96	9.49	0.34	3.13	0.72	n.a.	n.a.	5.07	4.96	3.46	0.66	1.57	2.51	8.75
100	100	Lipton	without	—	0.96	10.5	0.34	4.11	1.10	0.22	0.25	6.67	4.84	—	0.68	1.96	1.75	9.03
MEAN				0.82	0.97	9.49	0.34	3.40	0.87	0.20	0.23	5.50	2.69	4.71	0.74	1.95	2.18	10.0
100	200	Bristol	with	0.74	1.28	13.5	0.47	2.28	0.54	0.08	0.15	3.89	5.40	5.21	1.02	3.10	1.48	11.5
100	200	Bristol	with	0.77	1.11	9.59	0.29	3.65	0.67	0.19	0.18	5.76	3.18	5.01	0.69	2.68	2.29	11.2
100	200	Bristol	with	0.82	1.22	12.2	0.90	3.30	0.79	0.18	0.17	5.98	1.87	3.30	0.45	1.70	2.07	7.81
100	200	Bristol0.72	with	—	1.04	10.5	0.37	2.08	0.46	0.16	n.d.	3.40	3.57	—	0.95	4.10	2.64	17.2
MEAN				0.76	1.16	11.4	0.51	2.83	0.62	0.15	0.17	4.76	3.50	5.55	0.78	2.89	2.12	11.9
100	200	Bristol	without	—	1.34	8.40	0.55	2.54	0.45	0.16	n.d.	4.23	3.84	5.72	1.05	3.57	1.70	12.6
100	200	Bristol	without	0.72	1.25	10.4	0.30	3.26	0.58	0.27	0.09	5.08	3.49	5.97	1.00	2.75	1.68	11.7
100	200	Bristol	without	0.72	1.06	8.57	0.36	3.36	0.82	0.19	0.15	5.48	4.12	6.21	0.81	3.65	2.06	13.1
100	200	Bristol	without	—	1.36	13.8	0.47	3.16	0.70	0.19	0.30	5.37	2.47	—	0.79	3.38	1.96	14.2
MEAN				0.77	1.25	10.3	0.42	3.08	0.64	0.20	0.18	5.04	3.46	6.34	0.91	3.34	1.85	12.9

Table A.14: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ-glu-cys	GSH	P rog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
2002/2003																		
100	200	<i>Lipton</i>	with	0.57	0.91	9.33	0.45	2.35	0.66	0.07	0.14	4.01	2.89	5.16	0.74	2.70	1.82	10.9
100	200	<i>Lipton</i>	0.69 with		1.41	7.51	0.21	2.67	0.70	0.14	0.15	4.38	3.23	5.17	0.49	2.13	1.75	9.89
100	200	<i>Lipton</i>	0.66 with		1.28	21.8	1.32	2.53	0.73	0.11	n.d.	4.94	2.81	3.85	0.62	1.49	2.39	9.63
100	200	<i>Lipton</i>	with	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	5n.d.	—	0.56	2.06	1.82	10.1
MEAN				0.64	1.20	12.9	0.66	2.52	0.70	0.11	0.15	4.44	2.98	4.83	0.60	2.10	1.95	10.1
100	200	<i>Lipton</i>	w0.65ut	1.36	9.91	9.91	0.66	3.36	0.87	0.17	0.14	5.71	3.00	5.84	0.93	1.96	3.41	12.7
100	200	<i>Lipton</i>	w0.66ut	1.46	13.4	13.4	0.52	2.64	0.81	0.18	0.14	4.62	3.43	5.77	0.66	2.10	2.43	11.3
100	200	<i>Lipton</i>	w0.67ut	1.27	11.3	11.3	0.40	4.21	1.15	0.25	0.20	6.82	2.72	5.18	0.75	2.61	2.22	11.3
100	200	<i>Lipton</i>	w0.65ut	—	1.30	12.4	0.49	2.97	1.03	0.16	0.17	5.24	4.99	—	0.49	2.49	2.22	9.93
MEAN				0.68	1.35	11.8	0.52	3.29	0.97	0.19	0.16	5.60	3.04	5.33	0.70	2.29	2.57	11.3

note: n.a. – not available; m.v. – missing value; n.d. – not detected; cys – cysteine; γ-glu-cys – γ-glutamyl cysteine; GSH – glutathione; Prog – progoitrin; GBN – glucobrassicinapin; GN – gluconapin; GB – glucobrassicin; GNT – gluconasturtiin; GSL – total glucosinolates; Prot-S – S protein; 4-HGB – 4-hydroxy-glucobrassicin

Table A.15: Organic sulphur compounds in leaf and seed samples, Inverness, 2000-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Leaves										Seeds				
			Fungicide	Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB
2000/2001																	
0	100	Bristol	0.86 with	0.76	12.9	1.71	4.30	0.40	1.05	0.39	7.89	n.a.	2.63	0.61	1.75	n.d.	5.22
0	100	Bristol	with	0.63	22.9	0.84	2.19	0.21	0.52	0.22	4.02	1.36	0.82	0.11	0.57	n.d.	1.49
0	100	Bristol	with	0.52	11.0	1.24	4.04	0.43	0.62	0.32	6.67	1.73	1.24	0.15	0.73	n.d.	2.48
0	100	Bristol	0.64 with	0.35	17.6	0.90	2.59	0.26	0.32	0.25	4.35	1.32	0.89	0.08	0.45	n.d.	1.64
MEAN				0.79	16.1	1.17	3.28	0.32	0.63	0.30	5.73	1.47	1.39	0.23	0.88	n.d.	2.71
0	100	Bristol	without	0.83	8.53	0.64	2.49	0.39	0.23	0.39	4.16	1.87	1.42	0.22	0.77	n.d.	2.55
0	100	Bristol	without	1.11	7.21	0.64	2.55	0.26	0.42	0.15	4.03	2.79	1.70	0.31	0.93	0.01	3.23
0	100	Bristol	without	0.91	18.3	0.94	3.39	0.24	0.97	0.22	5.80	1.75	1.68	n.d.	0.18	0.27	2.75
0	100	Bristol	with	0.42	12.5	1.48	3.67	0.41	0.86	0.40	6.82	2.64	1.76	0.29	1.02	n.d.	3.50
MEAN				0.94	11.6	0.92	3.03	0.32	0.62	0.29	5.20	2.26	1.64	0.27	0.72	0.14	3.01
0	100	Lipton	0.67 with	0.48	16.6	1.04	2.81	0.59	0.19	0.30	4.96	n.a.	1.64	0.14	0.79	n.d.	2.70
0	100	Lipton	0.70 with	0.67	9.87	0.77	1.93	0.30	0.18	0.21	3.41	n.a.	1.65	0.13	0.95	0.02	2.82
0	100	Lipton	with	0.52	19.6	0.87	2.50	0.37	0.21	0.27	4.23	2.01	1.23	0.11	0.54	n.d.	2.29
0	100	Lipton	0.78 with	0.54	8.63	1.39	3.45	0.50	0.32	0.49	6.17	2.26	1.31	n.d.	0.67	n.d.	2.43
MEAN				0.75	13.7	1.02	2.67	0.44	0.23	0.32	4.69	2.13	1.45	0.13	0.74	0.02	2.56
0	100	Lipton	with	0.82	11.0	2.31	4.69	0.73	0.61	1.08	9.46	n.a.	1.18	0.11	0.43	n.d.	1.87
0	100	Lipton	with	1.26	9.53	2.20	4.84	0.71	0.46	0.39	8.62	n.a.	2.04	0.29	0.87	0.01	3.43
0	100	Lipton	without	0.38	13.4	0.89	2.54	0.34	0.60	0.31	4.70	2.13	2.00	0.28	0.99	n.d.	3.70
0	100	Lipton	with	0.54	10.8	2.56	5.35	0.91	0.71	0.47	10.0	1.78	2.13	0.32	1.15	n.d.	4.15
MEAN				0.95	11.2	1.99	4.36	0.67	0.60	0.56	8.21	1.95	1.84	0.25	0.86	0.01	3.29
0	200	Bristol	with	0.69	7.52	0.84	2.77	0.25	0.43	0.26	4.57	2.55	0.73	0.12	0.25	n.d.	1.15
0	200	Bristol	with	0.85	12.4	1.29	3.08	0.42	0.39	0.33	5.52	1.44	m.v.	m.v.	m.v.	n.d.	m.v.
0	200	Bristol	with	0.76	12.0	1.55	3.63	0.39	0.64	0.28	6.51	1.80	0.81	n.d.	0.31	n.d.	1.33
0	200	Bristol	0.77 with	0.56	7.20	0.64	2.61	0.21	0.55	0.24	4.26	n.a.	0.62	n.d.	0.45	n.d.	1.35
MEAN				0.77	9.80	1.08	3.02	0.32	0.50	0.28	5.21	1.93	0.72	0.12	0.33	n.d.	1.28
0	200	Bristol	without	0.47	7.35	1.44	3.88	0.32	0.61	0.42	6.69	2.09	0.34	0.06	0.38	n.d.	0.83
0	200	Bristol	with	0.79	8.04	1.03	3.61	0.30	0.74	0.29	5.41	n.a.	0.78	0.10	0.39	0.03	1.38
0	200	Bristol	without	0.77	19.1	1.32	4.23	0.30	0.71	0.30	6.88	2.27	0.91	0.10	0.60	n.d.	1.75
0	200	Bristol	with	0.60	9.03	1.23	3.39	0.31	0.73	0.33	6.01	2.74	1.21	0.15	0.64	n.d.	2.34
MEAN				0.73	10.9	1.25	3.78	0.31	0.70	0.33	6.25	2.37	0.81	0.10	0.50	0.03	1.57

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				Leaves				Seeds						
				γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL	
2000/2001																		
μmol g ⁻¹																		
0	200	Lipton	with	0.71	1.18	12.1	1.08	2.48	0.43	0.29	0.25	4.56	2.29	1.67	0.14	0.66	n.d.	2.55
0	200	Lipton	with	0.92	0.71	11.3	0.71	1.74	0.22	0.27	0.23	3.20	6.66	1.26	0.10	0.57	0.02	2.05
0	200	Lipton	with	0.85	0.70	11.7	1.48	3.46	0.42	0.33	0.31	6.02	n.a.	1.52	0.14	0.65	n.d.	2.63
0	200	Lipton	0.65 with	—	0.50	11.8	1.22	2.86	0.37	0.41	0.34	5.22	11.74	—	0.07	0.53	n.d.	2.14
MEAN				0.78	0.77	11.7	1.12	2.63	0.36	0.32	0.28	4.75	3.56	1.41	0.11	0.60	0.02	2.34
0	200	Lipton	without	0.79	0.61	10.0	1.77	3.74	0.49	0.60	1.13	7.75	n.a.	1.59	0.19	0.65	0.03	2.60
0	200	Lipton	without	0.76	0.97	12.1	1.90	4.03	0.50	0.50	0.35	7.29	n.a.	1.66	0.18	0.59	0.01	2.53
0	200	Lipton	without	0.73	0.65	8.39	1.40	3.21	0.43	0.37	0.35	5.78	3.07	2.05	0.14	0.86	0.06	3.36
0	200	Lipton	without	—	0.47	11.5	1.70	3.46	0.47	1.01	0.58	7.23	2.35	—	0.15	0.54	n.d.	2.41
MEAN				0.78	0.68	10.5	1.69	3.61	0.47	0.62	0.60	7.01	2.71	1.66	0.16	0.66	0.04	2.73
100	100	Bristol	with	1.42	1.20	7.66	2.03	4.79	0.70	0.91	0.61	9.07	0.85	3.41	0.70	2.22	0.01	6.69
100	100	Bristol	with	1.75	1.23	15.9	1.72	3.82	0.66	0.67	0.47	7.37	n.a.	2.31	0.35	1.59	0.04	4.50
100	100	Bristol	with	1.60	0.82	9.33	2.10	5.44	0.67	1.16	0.44	9.86	n.a.	3.19	0.65	2.02	n.d.	6.51
100	100	Bristol	0.62 with	—	0.63	14.1	1.88	4.66	0.73	0.53	0.44	8.27	3.22	—	0.63	2.14	n.d.	6.81
MEAN				1.60	0.97	11.7	1.93	4.68	0.69	0.82	0.49	8.64	0.85	3.03	0.58	1.99	0.03	6.13
100	100	Bristol	without	1.61	0.96	12.2	1.37	3.92	0.56	0.43	0.48	6.77	2.00	3.10	0.79	1.96	0.02	6.32
100	100	Bristol	without	1.60	1.21	8.39	2.94	6.24	0.91	1.15	0.37	11.6	n.a.	3.13	0.90	1.90	0.02	6.50
100	100	Bristol	without	1.38	0.86	11.0	2.85	6.28	0.79	1.09	0.49	11.5	3.73	3.46	0.87	2.08	n.d.	7.34
100	100	Bristol	without	—	0.66	12.1	1.11	3.81	0.50	0.52	0.33	6.28	2.16	—	1.08	2.02	n.d.	6.87
MEAN				1.51	0.92	10.9	2.07	5.06	0.69	0.80	0.42	9.06	2.64	3.21	0.91	1.99	0.02	6.76
100	100	Lipton	with	0.71	0.58	16.2	1.77	4.45	0.87	0.30	0.29	7.95	n.a.	5.17	0.90	2.90	0.02	9.41
100	100	Lipton	with	1.68	1.01	12.2	1.03	2.59	0.41	0.53	0.61	4.64	n.a.	1.98	0.34	1.05	0.02	3.61
100	100	Lipton	with	1.48	0.86	13.0	1.92	4.02	0.77	0.53	0.59	7.88	2.73	3.34	0.60	1.58	n.d.	6.16
100	100	Lipton	0.27 with	—	0.73	13.5	1.72	3.93	0.66	0.22	0.33	7.46	3.25	—	0.55	1.67	n.d.	6.25
MEAN				1.29	0.80	13.7	1.61	3.75	0.68	0.39	0.45	6.98	2.73	3.44	0.60	1.80	0.02	6.36
100	100	Lipton	without	1.28	0.95	12.1	2.45	5.47	1.10	0.89	0.47	10.7	n.a.	2.99	0.79	0.70	0.02	4.91
100	100	Lipton	without	1.44	1.33	10.0	2.45	5.03	0.94	0.73	0.42	9.81	n.a.	3.44	0.73	1.60	0.02	6.16
100	100	Lipton	without	1.50	1.11	9.50	1.85	4.63	0.69	1.10	0.55	8.34	n.a.	4.22	0.94	2.04	0.03	8.08
100	100	Lipton	without	—	0.78	14.0	2.52	5.18	1.01	0.69	0.94	10.4	3.95	—	0.90	1.87	n.d.	7.62
MEAN				1.44	1.04	11.4	2.32	5.07	0.94	0.85	0.60	9.80	1.91	3.62	0.84	1.55	0.03	6.69

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves							Seeds							
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL
μmol g ⁻¹																		
2000/2001																		
100	200	<i>Bristol</i>	with	1.62	1.09	17.8	1.75	3.63	0.60	0.56	0.38	7.14	1.48	4.08	0.71	2.37	0.01	7.54
100	200	<i>Bristol</i>	with	1.58	1.15	11.2	1.24	3.57	0.56	0.93	0.41	6.32	n.a.	3.54	0.58	2.08	0.06	6.56
100	200	<i>Bristol</i>	with	—	1.22	8.63	2.17	5.09	0.75	0.52	0.47	9.38	2.13	4.31	0.62	2.17	0.02	7.82
100	200	<i>Bristol</i>	with	—	0.79	10.1	1.76	3.73	0.63	0.64	0.51	7.14	0.96	—	0.50	1.70	n.d.	5.71
MEAN				1.46	1.06	11.9	1.73	4.01	0.64	0.66	0.44	7.49	1.32	3.75	0.60	2.08	0.03	6.91
100	200	<i>Bristol</i>	without	1.38	1.13	11.8	1.31	3.55	0.53	0.39	0.36	6.17	2.65	3.82	0.63	0.08	0.02	4.97
100	200	<i>Bristol</i>	without	1.87	1.22	13.9	2.25	4.52	0.66	1.01	0.41	8.88	3.84	3.25	0.63	1.58	0.02	5.82
100	200	<i>Bristol</i>	without	1.36	0.80	9.05	2.64	6.07	0.84	0.81	0.52	10.9	3.57	4.46	0.97	2.60	0.06	8.55
100	200	<i>Bristol</i>	without	—	0.93	11.7	1.76	4.26	0.59	0.76	0.79	8.19	0.95	—	0.85	2.08	n.d.	7.66
MEAN				1.51	1.02	11.6	1.99	4.60	0.66	0.74	0.52	8.54	2.63	3.88	0.77	1.58	0.03	6.75
100	200	<i>Lipton</i>	with	1.83	1.19	8.56	0.75	2.80	0.55	0.27	0.24	4.67	n.a.	3.91	0.59	1.89	0.02	6.67
100	200	<i>Lipton</i>	with	—	1.32	14.6	1.03	2.69	0.52	0.40	0.28	4.78	0.67	3.09	0.40	1.48	0.01	5.19
100	200	<i>Lipton</i>	with	—	0.94	14.1	1.20	3.30	0.56	0.51	0.36	5.76	n.a.	3.36	0.42	1.47	n.d.	5.83
100	200	<i>Lipton</i>	with	—	0.89	9.64	1.53	3.59	0.67	0.40	0.43	6.67	3.61	—	0.46	1.43	n.d.	6.31
MEAN				1.47	1.09	11.7	1.13	3.09	0.58	0.40	0.33	5.47	0.67	3.51	0.47	1.57	0.02	6.00
100	200	<i>Lipton</i>	without	—	0.75	11.0	2.19	4.42	0.85	0.70	0.41	8.59	n.a.	3.96	0.84	m.v.	0.02	5.46
100	200	<i>Lipton</i>	without	—	0.97	11.3	1.62	3.79	0.75	0.42	0.30	6.90	n.a.	4.12	0.88	1.79	0.02	7.25
100	200	<i>Lipton</i>	without	—	1.05	15.3	1.16	3.11	0.51	0.67	0.36	5.86	n.a.	4.16	0.78	1.74	n.d.	7.48
100	200	<i>Lipton</i>	without	—	0.77	11.2	1.77	4.03	0.81	0.65	0.33	7.61	4.24	—	0.84	1.89	n.d.	7.75
MEAN				1.40	0.89	12.2	1.69	3.83	0.73	0.61	0.35	7.24	n.a.	4.12	0.83	1.81	0.02	6.98
2001/2002																		
0	100	<i>Bristol</i>	with	0.88	0.28	16.3	0.35	2.69	0.64	0.12	0.29	4.10	2.59	2.36	0.46	1.39	n.d.	4.21
0	100	<i>Bristol</i>	with	—	0.25	7.46	0.10	1.34	0.29	0.04	0.17	1.94	3.11	1.47	0.16	0.87	n.d.	2.50
0	100	<i>Bristol</i>	with	—	0.10	12.6	0.19	1.49	0.31	0.08	0.14	2.28	1.88	3.04	0.40	1.62	n.d.	5.17
0	100	<i>Bristol</i>	with	—	0.52	22.9	0.17	2.04	0.27	0.12	0.22	2.92	1.96	—	0.30	1.03	n.d.	3.70
MEAN				0.62	0.29	14.8	0.20	1.89	0.38	0.09	0.20	2.81	2.38	2.20	0.33	1.23	n.d.	3.89
0	100	<i>Bristol</i>	without	—	0.33	19.7	0.12	1.30	0.28	0.05	n.d.	1.75	3.24	2.71	0.48	1.55	n.d.	4.75
0	100	<i>Bristol</i>	without	—	0.19	14.2	0.05	1.55	0.42	0.09	0.22	2.79	2.95	2.20	0.33	1.25	n.d.	4.00
0	100	<i>Bristol</i>	without	—	0.12	12.3	0.32	1.83	0.36	0.09	0.15	2.91	2.27	2.36	0.27	1.22	n.d.	4.16
0	100	<i>Bristol</i>	without	—	0.30	13.8	0.12	1.81	0.43	0.07	0.24	2.82	2.82	—	0.47	1.80	n.d.	5.72
MEAN				0.53	0.23	15.0	0.15	1.62	0.37	0.07	0.20	2.57	2.74	2.52	0.39	1.46	n.d.	4.66

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				γ-glu-cys				Leaves				Seeds			
				Prog	GSH	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL				
2001/2002																			
0	100	Lipton	with	0.61	0.39	19.9	1.00	4.47	0.88	0.26	0.43	7.08	2.99	1.91	0.31	0.86	n.d.	3.08	
0	100	Lipton	with	0.24	0.24	12.4	0.46	1.63	0.36	0.13	0.21	2.80	2.31	2.07	0.22	0.90	n.d.	3.44	
0	100	Lipton	with	0.11	0.24	15.4	0.41	1.60	0.37	0.09	0.16	2.85	2.43	2.46	0.36	1.18	n.d.	4.16	
0	100	Lipton	with	0.24	0.24	14.6	0.23	0.86	0.12	0.07	0.09	1.41	2.06	—	0.26	0.89	n.d.	3.75	
MEAN				0.49	0.24	15.6	0.52	2.14	0.43	0.14	0.22	3.54	2.53	2.13	0.29	0.96	n.d.	3.61	
0	100	Lipton	with	0.26	0.26	14.0	0.32	1.00	0.15	0.06	0.09	1.62	3.03	2.73	0.46	1.28	n.d.	4.46	
0	100	Lipton	with	0.25	0.25	19.2	0.62	2.45	0.46	0.12	0.17	3.81	2.23	3.14	0.46	1.44	n.d.	5.61	
0	100	Lipton	with	0.16	0.16	13.0	0.26	0.82	0.17	0.07	0.10	1.58	2.05	2.39	0.52	1.07	n.d.	4.13	
0	100	Lipton	with	0.14	0.14	7.61	0.50	1.66	0.42	0.11	0.18	2.94	2.90	—	0.27	0.76	n.d.	3.41	
MEAN				0.44	0.20	13.4	0.43	1.48	0.30	0.09	0.13	2.49	2.41	2.54	0.43	1.14	n.d.	4.40	
0	200	Bristol	with	0.68	m.v.	3.59	m.v.	m.v.	m.v.	m.v.	m.v.	m.v.	3.14	1.57	0.20	0.86	n.d.	2.63	
0	200	Bristol	with	0.23	0.23	4.30	0.10	0.65	0.18	0.03	0.05	1.04	3.01	0.95	0.11	0.51	n.d.	1.57	
0	200	Bristol	with	0.14	0.14	11.9	0.06	1.49	0.30	0.04	0.17	2.26	2.63	1.82	0.20	1.04	n.d.	3.31	
0	200	Bristol	with	0.30	0.30	10.8	0.05	0.53	0.08	0.02	0.05	0.80	2.30	—	0.17	0.81	n.d.	2.50	
MEAN				0.47	0.22	7.64	0.07	0.89	0.19	0.03	0.09	1.37	2.77	1.45	0.17	0.81	n.d.	2.50	
0	200	Bristol	with	0.28	0.28	12.4	0.21	1.82	0.43	0.08	0.16	2.70	2.92	1.39	0.17	0.80	n.d.	2.36	
0	200	Bristol	with	0.24	0.24	10.6	0.27	1.78	0.44	0.09	0.16	2.75	3.26	1.77	0.19	0.88	n.d.	3.04	
0	200	Bristol	with	0.16	0.16	11.1	0.23	1.74	0.38	0.06	0.20	2.78	2.58	1.24	0.17	0.63	n.d.	2.03	
0	200	Bristol	with	0.23	0.23	10.8	0.53	2.84	0.66	0.14	0.34	4.88	2.89	—	0.11	0.36	n.d.	1.42	
MEAN				0.51	0.23	11.2	0.31	2.04	0.48	0.09	0.21	3.28	2.77	1.30	0.16	0.67	n.d.	2.21	
0	200	Lipton	with	m.v.	m.v.	9.67	0.40	1.73	0.42	0.11	0.17	2.98	2.47	1.49	0.16	0.61	n.d.	2.26	
0	200	Lipton	with	0.21	0.21	6.80	0.16	0.47	0.09	0.03	0.05	0.79	3.04	1.16	0.12	0.46	n.d.	1.73	
0	200	Lipton	with	0.18	0.18	16.6	0.38	0.88	0.22	0.05	0.08	1.68	2.46	2.48	0.30	1.26	n.d.	4.21	
0	200	Lipton	with	0.31	0.31	8.07	0.12	1.19	0.25	0.07	0.11	1.80	2.90	—	0.09	0.37	n.d.	1.54	
MEAN				0.50	0.23	10.3	0.26	1.07	0.24	0.07	0.10	1.81	2.74	1.51	0.17	0.67	n.d.	2.44	
0	200	Lipton	with	0.20	0.20	8.0	0.33	1.33	0.33	0.05	0.11	2.15	3.37	2.10	0.26	0.95	n.d.	3.31	
0	200	Lipton	with	0.36	0.36	22.1	0.06	1.07	0.27	0.07	0.11	1.58	3.45	2.32	0.27	0.91	n.d.	3.81	
0	200	Lipton	with	0.17	0.17	15.2	0.46	1.19	0.25	0.07	0.10	2.19	2.48	1.51	0.13	0.74	n.d.	2.65	
0	200	Lipton	with	0.31	0.31	14.1	0.28	3.92	0.62	m.v.	m.v.	5.60	2.52	—	0.28	1.03	n.d.	4.46	
MEAN				0.44	0.26	14.8	0.28	1.88	0.37	0.06	0.11	2.88	2.92	2.11	0.24	0.91	n.d.	3.56	

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				Leaves				Seeds						
				γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL	
2001/2002																		
μmol g ⁻¹																		
100	100	<i>Bristol</i>	with	1.00	0.48	24.5	0.51	3.55	0.73	0.14	0.32	5.25	3.86	4.62	0.92	3.29	n.d.	8.83
100	100	<i>Bristol</i>	with	1.34	0.64	37.0	0.37	3.29	0.85	0.14	0.46	5.14	6.55	3.83	0.65	2.62	n.d.	7.13
100	100	<i>Bristol</i>	with	1.11	0.35	34.4	0.21	1.72	0.40	0.10	0.26	2.85	2.30	4.84	0.92	3.47	n.d.	9.61
100	100	<i>Bristol</i>	with	—	<u>0.87</u>	<u>37.5</u>	n.d.	<u>2.80</u>	<u>0.45</u>	<u>0.12</u>	<u>0.31</u>	<u>3.87</u>	<u>3.00</u>	—	<u>0.76</u>	<u>2.67</u>	<u>n.d.</u>	<u>7.92</u>
MEAN				1.21	0.58	33.4	0.36	2.84	0.61	0.13	0.34	4.28	3.93	4.30	0.81	3.01	n.d.	8.37
100	100	<i>Bristol</i>	without	1.43	0.96	36.3	0.42	2.98	0.76	0.16	n.d.	4.33	6.75	5.38	1.25	3.43	n.d.	10.2
100	100	<i>Bristol</i>	without	0.88	0.37	36.2	0.47	2.54	0.67	0.16	0.19	4.04	5.69	4.92	0.90	3.37	n.d.	9.59
100	100	<i>Bristol</i>	without	0.96	0.38	34.2	0.37	1.57	0.54	0.14	0.26	3.05	4.09	4.73	0.81	3.15	n.d.	9.20
100	100	<i>Bristol</i>	without	—	<u>0.42</u>	<u>29.6</u>	<u>0.11</u>	<u>2.26</u>	<u>0.27</u>	<u>0.14</u>	<u>0.26</u>	<u>3.10</u>	<u>4.07</u>	—	<u>1.01</u>	<u>3.00</u>	<u>n.d.</u>	<u>8.74</u>
MEAN				1.08	0.53	34.1	0.34	2.34	0.56	0.15	0.24	3.63	5.30	4.77	0.99	3.24	n.d.	9.42
100	100	<i>Lipton</i>	with	0.99	0.66	34.1	1.68	5.95	1.15	0.28	0.39	9.64	5.50	3.69	0.83	1.82	n.d.	6.34
100	100	<i>Lipton</i>	with	1.41	0.82	11.1	1.03	2.75	0.52	0.18	0.20	4.66	5.28	3.89	0.72	1.91	n.d.	6.84
100	100	<i>Lipton</i>	with	1.35	0.50	43.0	0.73	2.30	0.54	0.18	0.25	4.27	3.65	4.03	0.78	1.93	n.d.	7.75
100	100	<i>Lipton</i>	with	—	<u>0.87</u>	<u>11.4</u>	<u>0.26</u>	<u>3.18</u>	<u>0.46</u>	<u>0.29</u>	<u>0.25</u>	<u>4.54</u>	<u>3.93</u>	—	<u>0.65</u>	<u>1.74</u>	<u>n.d.</u>	<u>6.37</u>
MEAN				1.26	0.71	24.9	0.92	3.54	0.67	0.23	0.27	5.78	4.97	3.74	0.74	1.85	n.d.	6.83
100	100	<i>Lipton</i>	without	1.41	0.72	8.27	0.67	1.71	0.38	0.12	0.17	3.06	7.86	3.77	0.80	1.84	n.d.	6.41
100	100	<i>Lipton</i>	without	1.12	0.48	43.8	1.55	3.63	0.74	0.28	0.28	6.64	4.90	4.91	0.99	2.25	n.d.	8.60
100	100	<i>Lipton</i>	without	0.84	0.40	37.2	0.65	2.39	0.41	0.11	0.15	3.87	4.72	4.56	0.95	2.33	n.d.	8.08
100	100	<i>Lipton</i>	without	—	<u>0.38</u>	<u>32.6</u>	<u>0.41</u>	<u>1.37</u>	<u>0.31</u>	<u>0.10</u>	<u>0.14</u>	<u>2.39</u>	<u>4.70</u>	—	<u>0.90</u>	<u>2.20</u>	<u>n.d.</u>	<u>8.50</u>
MEAN				1.12	0.49	30.5	0.82	2.28	0.46	0.15	0.18	3.99	5.56	4.49	0.91	2.15	n.d.	7.90
100	200	<i>Bristol</i>	with	0.11	0.10	3.60	0.24	2.13	0.54	0.15	0.20	3.28	6.67	5.56	0.83	3.16	n.d.	9.55
100	200	<i>Bristol</i>	with	1.63	1.39	15.8	0.18	1.52	0.45	0.11	0.21	2.46	5.99	5.97	0.87	3.80	n.d.	10.6
100	200	<i>Bristol</i>	with	1.02	0.32	33.8	0.16	1.17	0.28	0.06	0.13	1.87	6.12	7.13	1.17	4.19	n.d.	13.0
100	200	<i>Bristol</i>	with	—	<u>1.13</u>	<u>31.1</u>	<u>0.20</u>	<u>2.97</u>	<u>0.45</u>	<u>0.19</u>	<u>0.42</u>	<u>4.38</u>	<u>5.37</u>	—	<u>0.92</u>	<u>3.04</u>	<u>n.d.</u>	<u>10.0</u>
MEAN				1.04	0.74	21.1	0.20	1.95	0.43	0.13	0.24	3.00	6.26	5.98	0.95	3.55	n.d.	10.8
100	200	<i>Bristol</i>	without	1.62	1.50	11.9	0.93	n.d.	2.14	0.59	0.96	4.68	5.71	6.67	1.37	4.23	n.d.	12.4
100	200	<i>Bristol</i>	without	1.60	1.24	47.3	0.24	3.42	1.01	0.21	0.42	5.33	4.95	6.37	1.01	3.70	n.d.	11.1
100	200	<i>Bristol</i>	without	1.14	0.67	40.6	0.23	1.13	0.13	0.10	0.03	1.80	4.70	6.37	0.94	3.60	n.d.	11.3
100	200	<i>Bristol</i>	without	—	<u>0.86</u>	<u>48.2</u>	<u>0.36</u>	<u>3.02</u>	<u>0.82</u>	<u>0.18</u>	<u>0.31</u>	<u>4.92</u>	<u>3.93</u>	—	<u>0.90</u>	<u>3.56</u>	<u>n.d.</u>	<u>11.3</u>
MEAN				1.35	1.07	37.0	0.44	2.52	1.03	0.27	0.43	4.18	4.67	6.34	1.05	3.77	n.d.	11.5

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves										Seeds				
				Cys	γ-glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	P rog	GBN	GN	4-HGB	GSL
2001/2002																		
100	200	<i>Lipton</i>	with	2.45	1.97	19.5	0.80	3.11	0.57	0.19	0.26	4.95	4.08	5.92	0.94	2.65	n.d.	9.51
100	200	<i>Lipton</i>	with	1.50	1.27	14.9	0.83	2.79	0.65	0.29	0.30	4.88	5.85	5.91	0.76	2.53	n.d.	9.54
100	200	<i>Lipton</i>	with	0.87	0.39	42.0	0.45	1.66	0.37	0.09	0.14	2.98	5.36	6.37	0.97	2.87	n.d.	10.6
100	200	<i>Lipton</i>	with	<u>1.38</u>	<u>1.25</u>	<u>21.2</u>	<u>0.32</u>	<u>2.68</u>	<u>0.57</u>	<u>0.17</u>	<u>0.23</u>	<u>4.09</u>	<u>4.39</u>	<u>5.74</u>	<u>0.80</u>	<u>2.03</u>	<u>n.d.</u>	<u>8.18</u>
MEAN				1.58	1.25	24.4	0.60	2.56	0.54	0.18	0.23	4.23	5.17	5.74	0.87	2.52	n.d.	9.45
100	200	<i>Lipton</i>	without	1.63	1.54	15.3	0.47	1.64	0.45	0.21	0.20	3.08	5.65	6.48	1.09	2.89	n.d.	10.5
100	200	<i>Lipton</i>	without	0.88	0.56	47.9	0.87	2.38	0.57	0.20	0.22	4.45	4.77	6.02	0.92	2.51	n.d.	10.4
100	200	<i>Lipton</i>	without	0.97	0.59	40.6	0.83	2.11	0.46	0.14	0.17	3.93	3.69	6.51	0.77	3.06	n.d.	11.0
100	200	<i>Lipton</i>	without	<u>1.22</u>	<u>1.22</u>	<u>51.3</u>	<u>0.21</u>	<u>4.37</u>	<u>0.67</u>	<u>m.v.</u>	<u>0.32</u>	<u>6.52</u>	<u>5.84</u>	<u>6.34</u>	<u>0.93</u>	<u>2.82</u>	<u>n.d.</u>	<u>10.6</u>
MEAN				1.37	0.98	38.8	0.60	2.63	0.54	0.18	0.23	4.50	4.99	6.34	0.93	2.82	n.d.	10.6
2002/2003																		
0	100	<i>Bristol</i>	with	0.16	0.71	12.3	0.91	1.01	0.36	0.12	0.21	3.01	1.08	3.22	0.43	2.31	1.89	8.59
0	100	<i>Bristol</i>	with	0.53	0.77	18.8	0.56	1.23	0.54	0.16	0.35	2.97	1.17	3.01	0.43	2.41	1.60	8.22
0	100	<i>Bristol</i>	with	0.84	0.67	16.9	0.80	0.37	0.22	0.07	0.13	1.70	0.59	3.30	0.38	2.47	1.24	8.14
0	100	<i>Bristol</i>	with	<u>0.85</u>	<u>0.75</u>	<u>7.92</u>	<u>0.15</u>	<u>0.61</u>	<u>0.15</u>	<u>0.03</u>	<u>0.07</u>	<u>1.16</u>	<u>3.40</u>	<u>3.31</u>	<u>0.49</u>	<u>1.65</u>	<u>1.64</u>	<u>7.97</u>
MEAN				0.65	0.75	14.0	0.61	0.81	0.32	0.09	0.19	2.21	1.06	3.31	0.43	2.21	1.59	8.23
0	100	<i>Bristol</i>	without	0.34	0.99	9.57	0.69	2.79	0.99	0.19	0.40	5.72	1.40	3.93	0.47	2.54	1.48	9.04
0	100	<i>Bristol</i>	without	0.81	0.91	8.46	0.95	1.93	1.15	0.19	0.56	4.87	n.a.	3.37	0.46	2.37	2.00	8.75
0	100	<i>Bristol</i>	without	0.89	0.65	18.6	0.64	0.32	0.33	0.03	0.09	1.40	0.18	3.85	0.44	2.33	1.70	8.89
0	100	<i>Bristol</i>	without	<u>0.70</u>	<u>0.70</u>	<u>16.5</u>	<u>0.30</u>	<u>0.62</u>	<u>0.19</u>	<u>0.05</u>	<u>0.05</u>	<u>1.33</u>	<u>0.09</u>	<u>3.71</u>	<u>0.60</u>	<u>2.42</u>	<u>1.91</u>	<u>9.34</u>
MEAN				0.73	0.81	13.3	0.64	1.42	0.66	0.11	0.27	3.33	0.56	3.71	0.50	2.41	1.77	9.00
0	100	<i>Lipton</i>	with	0.33	0.74	15.5	0.90	1.33	0.59	0.16	0.18	3.62	0.80	3.05	0.33	1.71	1.30	6.79
0	100	<i>Lipton</i>	with	0.42	0.69	17.2	0.75	0.71	0.57	0.16	n.a.	2.20	1.27	3.19	0.31	2.01	2.11	8.03
0	100	<i>Lipton</i>	with	0.58	0.70	17.0	0.80	1.05	0.57	0.22	n.a.	2.85	0.42	3.82	0.56	2.34	1.68	8.88
0	100	<i>Lipton</i>	with	<u>0.52</u>	<u>0.71</u>	<u>16.6</u>	<u>0.82</u>	<u>1.03</u>	<u>0.58</u>	<u>0.18</u>	<u>0.06</u>	<u>2.89</u>	<u>3.64</u>	<u>3.42</u>	<u>0.37</u>	<u>1.37</u>	<u>2.81</u>	<u>8.46</u>
MEAN				0.52	0.71	16.6	0.82	1.03	0.58	0.18	0.12	2.89	0.95	3.42	0.39	1.86	1.97	8.04
0	100	<i>Lipton</i>	without	0.24	0.78	14.9	0.74	1.98	1.02	0.23	0.24	4.47	1.06	4.00	0.38	1.81	1.76	8.46
0	100	<i>Lipton</i>	without	0.77	0.64	15.6	0.69	0.57	0.46	0.07	0.08	1.96	1.33	2.96	0.37	1.61	1.90	7.12
0	100	<i>Lipton</i>	without	0.57	0.98	10.3	1.62	1.54	1.54	0.28	0.10	5.13	0.40	5.36	0.39	1.85	1.71	9.59
0	100	<i>Lipton</i>	without	<u>0.97</u>	<u>0.97</u>	<u>9.48</u>	<u>0.35</u>	<u>0.62</u>	<u>0.24</u>	<u>0.04</u>	<u>0.10</u>	<u>1.44</u>	<u>4.00</u>	<u>4.08</u>	<u>0.38</u>	<u>2.12</u>	<u>3.58</u>	<u>10.6</u>
MEAN				0.58	0.84	12.6	0.85	1.18	0.81	0.16	0.13	3.25	1.01	4.08	0.38	1.85	2.24	8.93

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cys				γ-glu-cys				Leaves				Seeds			
				GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB	GSL			
2002/2003																			
0	200	<i>Bristol</i>	with	0.42	1.11	9.65	0.72	0.58	0.21	0.08	0.17	2.14	3.39	1.45	0.18	1.10	0.87	3.91	
0	200	<i>Bristol</i>	with	0.37	1.01	18.7	0.42	0.58	0.22	0.09	0.13	1.58	2.47	1.77	0.22	1.31	1.43	5.11	
0	200	<i>Bristol</i>	with	0.47	1.14	19.2	0.71	0.76	0.38	0.27	0.31	2.59	2.24	3.32	0.37	2.10	1.01	7.38	
0	200	<i>Bristol</i>	with	—	0.99	9.94	0.70	0.52	0.12	0.07	0.12	1.59	1.69	—	0.20	0.67	1.04	3.92	
MEAN				0.46	1.06	14.4	0.64	0.61	0.23	0.13	0.18	1.98	2.47	2.06	0.24	1.29	1.09	5.08	
0	200	<i>Bristol</i>	without	0.12	0.89	12.0	0.67	0.91	0.29	0.15	0.12	2.37	2.95	3.26	0.39	2.10	1.71	7.93	
0	200	<i>Bristol</i>	without	0.63	1.56	19.8	0.77	1.72	1.29	0.20	0.56	4.64	1.44	2.22	0.32	1.26	1.28	5.43	
0	200	<i>Bristol</i>	without	0.70	0.85	9.44	0.70	0.27	0.21	0.06	n.a.	1.24	2.03	3.19	0.34	1.45	2.05	7.40	
0	200	<i>Bristol</i>	without	—	0.92	17.9	0.35	0.68	0.25	0.12	0.19	1.80	2.82	—	0.34	1.76	1.67	7.06	
MEAN				0.50	1.06	14.8	0.62	0.89	0.51	0.13	0.29	2.51	2.14	2.87	0.35	1.64	1.68	6.96	
0	200	<i>Lipton</i>	with	0.17	0.79	12.5	0.73	0.35	0.15	0.15	0.05	1.67	2.19	2.93	0.26	1.62	1.58	6.75	
0	200	<i>Lipton</i>	with	0.54	1.15	8.65	0.46	0.60	0.40	0.05	0.15	1.82	1.84	3.20	0.27	1.63	2.05	7.52	
0	200	<i>Lipton</i>	with	0.45	1.02	16.3	0.77	0.21	n.d.	0.08	n.a.	1.18	1.56	4.00	0.31	1.74	2.89	9.35	
0	200	<i>Lipton</i>	with	—	1.38	18.7	0.28	0.64	0.28	0.06	0.14	1.57	4.00	—	0.35	1.67	1.85	8.29	
MEAN				0.40	1.08	14.0	0.56	0.45	0.28	0.09	0.12	1.56	1.68	3.53	0.30	1.67	2.09	7.98	
0	200	<i>Lipton</i>	without	0.26	1.26	16.1	0.83	0.70	0.32	0.17	n.a.	2.37	1.71	4.26	0.27	1.82	1.56	8.26	
0	200	<i>Lipton</i>	without	0.55	0.97	17.7	0.64	0.25	0.27	0.07	n.a.	1.23	1.51	2.20	0.21	0.86	1.65	5.28	
0	200	<i>Lipton</i>	without	0.54	1.11	11.6	0.71	0.66	0.48	0.16	n.a.	2.07	1.62	3.44	0.32	1.58	2.19	7.82	
0	200	<i>Lipton</i>	without	—	1.18	9.80	0.66	1.14	0.47	0.14	0.24	2.96	2.59	—	0.36	1.72	1.41	7.51	
MEAN				0.46	1.13	13.8	0.71	0.69	0.39	0.13	0.24	2.16	1.84	3.40	0.29	1.50	1.70	7.22	
100	100	<i>Bristol</i>	with	0.43	0.89	8.36	0.82	1.61	0.61	0.19	0.34	3.97	1.24	3.82	0.44	2.58	1.90	9.46	
100	100	<i>Bristol</i>	with	0.71	1.16	7.94	0.72	1.17	0.49	0.16	0.21	2.90	1.07	3.16	0.40	2.79	0.75	7.71	
100	100	<i>Bristol</i>	with	1.03	0.96	9.48	0.88	0.50	n.d.	0.11	0.19	1.68	n.a.	4.16	0.52	2.82	1.70	9.86	
100	100	<i>Bristol</i>	with	—	0.94	8.03	0.15	1.10	0.39	0.08	0.09	2.06	2.82	—	0.77	2.43	2.26	10.8	
MEAN				0.82	0.99	8.45	0.64	1.10	0.50	0.13	0.21	2.65	1.76	3.99	0.53	2.66	1.65	9.46	
100	100	<i>Bristol</i>	without	0.37	0.83	16.6	0.71	2.28	1.01	0.32	0.21	4.94	2.09	4.20	0.58	3.11	1.23	9.78	
100	100	<i>Bristol</i>	without	1.07	1.18	8.29	0.79	2.67	1.51	0.26	0.71	6.26	n.a.	4.67	0.70	2.96	1.68	10.6	
100	100	<i>Bristol</i>	without	0.74	1.04	17.2	0.65	1.65	0.95	0.24	0.39	4.03	1.10	4.22	0.53	2.38	2.64	10.4	
100	100	<i>Bristol</i>	without	—	1.21	8.61	0.39	0.74	0.32	0.04	0.12	1.80	4.26	—	0.57	2.39	1.57	9.40	
MEAN				0.86	1.06	12.7	0.64	1.84	0.95	0.22	0.36	4.26	1.25	4.36	0.60	2.71	1.78	10.1	

Table A.15: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Leaves					Seeds								
				Cys	γ -glu-cys	GSH	Prog	GBN	GN	GB	GNT	GSL	Prot-S	Prog	GBN	GN	4-HGB
2002/2003																	
100	100	Lipton 53	with	0.84	18.5	0.69	1.68	0.70	0.22	0.18	3.85	1.24	3.94	0.40	1.98	1.97	8.80
100	100	Lipton	with	0.68	17.0	0.84	0.90	0.49	0.32	0.11	3.11	1.50	4.31	0.46	2.45	2.47	10.2
100	100	Lipton	with	0.79	8.15	0.65	0.37	0.36	0.09	0.11	1.74	0.50	4.39	0.40	1.94	1.20	8.46
100	100	Lipton 02	with	0.56	16.2	0.21	0.58	0.24	0.03	0.05	1.22	1.22	—	0.64	1.41	2.20	8.71
MEAN				0.76	14.9	0.60	0.88	0.45	0.16	0.11	2.48	1.11	4.23	0.47	1.94	1.96	9.06
100	100	Lipton	without	0.49	17.5	0.80	2.11	0.68	0.21	0.33	4.64	n.a.	3.91	0.39	1.65	3.14	9.59
100	100	Lipton	without	0.98	8.96	0.52	0.72	0.60	0.04	0.14	2.02	1.21	4.13	0.52	2.11	1.73	8.88
100	100	Lipton	without	0.90	11.1	0.75	0.60	0.49	0.09	n.a.	1.93	0.32	3.57	0.41	1.43	1.59	7.28
100	100	Lipton	without	1.22	7.61	0.90	1.70	0.68	0.11	0.24	3.98	1.60	—	0.62	2.48	2.40	12.4
MEAN				0.85	11.3	0.74	1.28	0.61	0.11	0.24	3.15	1.06	4.48	0.48	1.92	2.22	9.53
100	200	Bristol	with	0.40	8.90	0.81	1.45	0.51	0.22	0.19	3.70	2.19	4.10	0.55	2.97	1.84	10.1
100	200	Bristol	with	0.47	9.24	0.69	1.33	0.54	0.17	0.23	3.35	0.50	4.30	0.62	2.94	1.90	10.5
100	200	Bristol	with	0.72	13.5	0.72	1.25	0.70	0.26	0.34	3.39	0.22	4.94	0.70	3.60	2.27	12.2
100	200	Bristol 06	with	1.18	8.99	0.45	0.97	0.34	0.08	0.19	2.36	1.09	—	0.90	2.86	1.67	11.3
MEAN				0.61	10.2	0.67	1.25	0.52	0.18	0.24	3.20	1.00	4.61	0.69	3.09	1.92	11.0
100	200	Bristol	without	0.38	9.9	0.79	1.59	0.56	0.19	0.34	3.94	1.58	4.33	0.58	2.80	1.92	10.2
100	200	Bristol	without	0.80	12.9	0.57	0.65	0.49	0.04	0.22	2.04	1.75	4.85	0.66	2.80	1.93	10.8
100	200	Bristol	without	0.98	7.25	0.87	1.35	1.14	0.17	0.53	4.29	n.a.	5.96	0.89	3.15	1.97	12.6
100	200	Bristol	without	1.20	7.72	1.39	2.34	0.75	0.52	0.54	6.28	1.38	—	1.02	3.10	1.30	12.0
MEAN				0.74	9.46	0.91	1.48	0.74	0.23	0.41	4.14	1.55	5.23	0.79	2.96	1.78	11.4
100	200	Lipton	with	0.43	10.1	0.91	1.13	0.58	0.22	0.19	3.50	1.73	4.54	0.44	2.39	1.84	9.84
100	200	Lipton 068	with	1.21	8.87	0.43	0.55	0.37	0.15	n.a.	1.64	1.97	4.29	0.44	2.14	2.07	9.39
100	200	Lipton 067	with	1.10	10.2	0.68	0.21	0.20	0.06	n.a.	1.32	1.36	5.12	0.41	2.23	2.42	10.5
100	200	Lipton 062	with	1.07	10.3	n.d.	0.65	0.32	0.06	0.15	1.31	2.92	—	0.62	1.35	2.67	10.2
MEAN				0.60	9.86	0.67	0.63	0.37	0.12	0.17	1.94	1.99	4.72	0.48	2.03	2.25	9.98
100	200	Lipton	without	0.33	7.91	0.61	1.29	0.57	0.29	0.16	3.14	1.59	4.50	0.52	2.22	2.01	9.62
100	200	Lipton	without	1.10	8.19	0.69	0.34	0.42	0.05	n.a.	1.50	1.18	6.76	0.51	1.78	2.43	12.0
100	200	Lipton	without	0.79	7.67	0.77	1.33	0.94	0.20	0.28	3.61	0.74	5.11	0.57	2.23	2.35	10.8
100	200	Lipton	without	1.30	7.41	0.99	1.65	0.72	0.16	0.18	4.05	1.89	—	0.58	2.32	2.10	11.3
MEAN				0.70	7.80	0.76	1.15	0.66	0.18	0.21	3.08	1.20	5.57	0.55	2.13	2.22	10.9

note: n.a. – not available; m.v. m – missing value; n.d. – not detected; cys – cysteine; γ -glu-cys – γ -glutamyl cysteine; GSH – glutathione; Prog – progoinin; GBN – glucobrassicin; GN – gluconapin; GB – gluconapin; GNT – gluconasturtin; GSL – total glucosinolates; Prot-S – S protein; 4-HGB – 4-hydroxy-glucobrassicin

Table A.16: Results from the infection of winter oilseed rape by *Pyrenopeziza brassicae*, Braunschweig, 2001/2002.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		γ-glu-cys		Glutathione		LCD		OAS-TL	
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected
				H									
0	100	Bristol	with	1.40	0.53	0.96	1.01	13.7	10.3	14.6	8.96	1443.8	684.9
0	100	Bristol08	with		0.45	0.93	0.63	8.66	9.46	13.2	8.50	639.0	2085.7
0	100	Bristol13	with		0.39	0.66	1.23	10.8	8.02	19.5	7.98	3555.7	2423.5
0	100	Bristol85	with		0.42	0.59	0.77	8.90	8.23	16.9	18.794		1108.7
MEAN					0.45	0.79	0.91	10.5	9.00	16.0	7.85	1877.4	1575.7
0	100	Bristol	with		0.55	0.99	0.75	12.4	10.2	14.3	11.2	1971.7	1006.3
0	100	Bristol	with		0.64	m.v.	1.04	21.0	16.4	13.3	9.37	773.8	2428.3
0	100	Bristol	with		0.31	1.81	0.74	14.0	8.59	22.9	14.3	3506.4	3422.5
0	100	Bristol	with		0.42	0.94	1.26	9.64	10.1	17.4	2800.6	2028.4	2028.4
MEAN					0.48	1.25	0.95	14.3	11.34	17.0	12.1	2263.0	2221.4
0	100	Lipton16	with		0.32	0.87	1.10	14.8	7.37	13.1	12.2	1552.8	1170.1
0	100	Lipton74	with		0.82	1.25	3.42	11.6	13.8	16.8	9.28	1031.2	2152.9
0	100	Lipton19	with		1.11	0.94	5.40	13.5	10.3	19.5	9.36	2868.8	2537.2
0	100	Lipton04	with		0.52	0.89	1.55	12.3	15.6	17.8	2600.8	2163.3	2163.3
MEAN					0.69	0.99	2.87	13.1	11.8	16.8	11.2	2013.2	2005.9
0	100	Lipton	with		0.35	0.70	0.82	18.4	7.52	18.1	13.2	2618.7	1330.2
0	100	Lipton	with		0.30	0.89	0.72	13.4	7.85	16.5	8.60	1254.2	2196.5
0	100	Lipton	with		0.26	0.60	0.58	10.9	7.74	19.7	10.0	3692.1	2842.8
0	100	Lipton	with		0.26	0.85	0.94	11.8	9.52	19.3	2976.7	1781.9	1781.9
MEAN					0.29	0.76	0.76	13.6	8.16	18.4	11.4	2635.3	2037.9
0	200	Bristol69	with		0.59	0.96	1.46	15.5	15.2	16.8	15.1	2336.8	1256.8
0	200	Bristol01	with		0.42	0.82	1.73	14.4	12.4	18.9	12.9	814.4	3974.5
0	200	Bristol97	with		0.31	0.88	0.82	9.71	6.54	20.3	19.0	4389.4	3289.8
0	200	Bristol74	with		0.31	0.55	0.97	17.0	6.98	20.6	3179.0	2112.9	2112.9
MEAN					0.41	0.80	1.24	14.1	10.3	19.2	15.5	2680.1	2658.5
0	200	Bristol	with		0.37	m.v.	1.11	22.3	6.91	17.2	14.4	2295.7	1516.6
0	200	Bristol	with		0.45	0.85	0.28	9.19	12.6	16.4	11.8	1263.5	3454.8
0	200	Bristol	with		0.32	0.87	0.59	11.7	6.45	25.1	15.5	4284.7	3220.5
0	200	Bristol	with		0.42	1.01	1.03	10.4	12.4	15.9	3021.0	2447.2	2447.2
MEAN					0.39	0.91	0.75	13.4	9.58	18.7	14.2	2716.6	2659.8

Table A.16: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		γ -glu-cys		Glutathione		LCD		OAS-TL	
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected
				H		μ mol g ⁻¹		μ mol (mg prot x min) ⁻¹		μ mol (mg prot x min) ⁻¹		cys [nmol(mg prot x min) ⁻¹]	
0	200	Lipton	with	1.10	0.44	1.20	1.14	14.7	11.3	17.5	12.5	2339.1	1591.3
0	200	Lipton81	with		0.45	0.99	2.38	11.2	20.1	17.2	14.0	1177.6	2794.6
0	200	Lipton00	with		0.26	1.48	0.98	14.6	10.4	23.1	17.3	3558.5	4791.1
0	200	Lipton97	with		0.48	0.91	1.62	13.2	17.0	20.7	3074.7	2310.9	
MEAN					0.41	1.15	1.53	13.4	14.7	19.6	14.5	2537.4	2872.0
0	200	Lipton	with		0.51	0.51	1.39	13.6	16.8	17.6	14.4	2068.6	1223.7
0	200	Lipton	with		0.33	1.23	1.13	14.9	10.1	20.1	13.9	1491.6	3949.2
0	200	Lipton	with		0.27	1.02	0.83	14.9	7.86	28.9	17.7	4787.5	4372.5
0	200	Lipton	with		1.01	0.69	3.61	10.0	16.2	18.6	3486.8	2643.5	
MEAN					0.53	0.86	1.74	13.4	12.8	21.3	15.7	2957.3	3047.2
150	100	Bristol89	with		0.44	1.43	0.58	16.3	7.93	15.0	8.58	1159.2	428.7
150	100	Bristol03	with		0.43	2.10	0.67	15.2	6.12	11.9	9.08	763.7	2104.9
150	100	Bristol84	with		0.53	0.77	0.51	9.39	7.35	16.9	7.51	2586.9	2531.6
150	100	Bristol22	with		0.41	0.80	0.63	8.68	8.25	12.9	2082.6	1538.4	
MEAN					0.45	1.28	0.60	12.4	7.41	14.2	8.29	1638.1	1650.9
150	100	Bristol	with		0.63	1.22	0.84	16.1	11.4	10.8	10.2	1324.5	664.8
150	100	Bristol	with		0.63	3.14	0.42	15.0	12.0	12.1	8.01	686.6	1907.3
150	100	Bristol	with		2.37	1.73	m.v.	15.9	18.1	14.4	9.77	2416.1	2489.0
150	100	Bristol	with		0.42	0.86	0.78	8.65	6.69	10.1	2397.3	1368.0	
MEAN					1.01	1.74	0.68	13.9	12.1	11.9	9.43	1706.1	1607.3
150	100	Lipton58	with		0.50	1.28	0.19	17.0	11.0	15.5	9.21	1432.6	555.5
150	100	Lipton17	with		0.50	1.40	1.50	13.0	9.67	17.2	9.79	962.2	2338.8
150	100	Lipton57	with		0.33	2.54	0.76	23.9	7.22	11.5	7.38	3115.9	2398.1
150	100	Lipton80	with		0.44	1.57	0.62	15.2	8.51	13.3	2256.6	1350.2	
MEAN					0.44	1.70	0.77	17.3	9.11	14.4	8.51	1945.1	1660.7
150	100	Lipton	with		0.44	0.69	1.06	16.0	10.5	13.1	7.83	1396.8	484.2
150	100	Lipton	with		0.97	2.86	3.34	23.4	9.84	13.5	8.97	922.2	2536.0
150	100	Lipton	with		0.42	1.11	0.61	15.2	7.89	19.9	11.8	3004.8	2996.8
150	100	Lipton	with		1.90	1.46	3.03	14.2	18.9	14.9	1397.0	1540.5	
MEAN					0.94	1.53	2.01	17.2	11.8	15.4	9.90	1680.3	1889.4

Table A.16: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		γ-glu-cys		Glutathione		LCD		OAS-TL	
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected
				H						₂ S [nmol(mg prot x min) ⁻¹]		cys [nmol(mg prot x min) ⁻¹]	
150	200	<i>Bristol</i>	with	1.97	0.72	1.09	1.13	15.1	15.0	21.4	10.9	2078.6	850.4
150	200	<i>Bristol</i>	81 with		0.81	1.70	1.11	13.5	11.0	18.5	11.5	912.7	3216.0
150	200	<i>Bristol</i>	83 with		0.81	1.23	0.91	17.4	10.7	23.4	8.61	4108.6	2581.9
150	200	<i>Bristol</i>	21 with		0.62	0.81	0.99	8.69	11.6	16.7	2599.2		2127.9
MEAN					0.74	1.21	1.03	13.7	12.1	20.0	10.3	2423.6	2194.1
150	200	<i>Bristol</i>	with		0.86	1.18	1.03	15.9	15.5	15.0	9.87	1834.5	744.5
150	200	<i>Bristol</i>	with		0.62	3.24	1.54	16.9	11.6	15.3	11.7	1025.4	3530.1
150	200	<i>Bristol</i>	with		0.71	1.06	1.55	13.8	15.3	18.3	15.9	3818.1	4205.2
150	200	<i>Bristol</i>	with		0.95	4.55	1.22	22.4	16.9	19.8	3009.8		2506.8
MEAN					0.79	2.51	1.33	17.2	14.9	17.1	12.7	2422.0	2746.7
150	200	<i>Lipton</i>	69 with		0.88	0.63	1.32	15.2	17.5	19.7	10.1	1824.5	1059.0
150	200	<i>Lipton</i>	94 with		0.44	2.90	1.27	17.4	9.56	15.5	11.2	946.0	2921.9
150	200	<i>Lipton</i>	201 with		0.45	2.61	0.87	17.6	8.51	21.2	14.1	4402.9	3606.7
150	200	<i>Lipton</i>	291 with		0.49	1.58	0.99	16.0	10.8	17.8	2699.6		2179.2
MEAN					0.57	1.93	1.11	16.6	11.6	18.6	12.3	2468.3	2441.7
150	200	<i>Lipton</i>	with		1.02	1.99	1.82	17.3	12.9	15.8	11.6	1963.3	999.4
150	200	<i>Lipton</i>	with		0.54	2.65	1.42	19.7	12.0	17.5	11.5	1055.1	2864.4
150	200	<i>Lipton</i>	with		0.48	2.59	0.92	18.6	10.8	26.3	12.0	4026.8	3212.1
150	200	<i>Lipton</i>	with		0.74	1.47	0.95	16.3	16.6	18.5	2973.3		2382.2
MEAN					0.69	2.17	1.28	18.0	13.0	19.5	12.1	2505.2	2364.5

note: m. v. – missing value; γ-glu-cys – γ-glutamyl-cysteine; LCD – L-cysteine desulphhydrase; OAS-TL – O-acetylserine(thio)lyase

Table A.17: Results from the infection of winter oilseed rape by *Pyrenopeziza brassicae*, Aberdeen, 2001-2003.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		Glutathione		Cysteine		Glutathione	
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected
				2001/2002				2002/2003			
				μmol g ⁻¹							
0	100	<i>Bristol</i>	with	0.06	0.99	0.19	9.44	1.31	0.95	11.3	17.0
0	100	<i>Bristol</i>	with		0.89	0.41	12.4	0.98	0.84	20.4	15.8
0	100	<i>Bristol</i>	with		0.69	16.7	11.8	1.49	0.85	9.35	15.2
0	100	<i>Bristol</i>	with		0.86	18.8	14.0	1.76	1.12	14.4	15.6
MEAN					0.85	9.02	11.9	1.38	0.94	13.8	15.9
0	100	<i>Bristol</i>	0.04without		0.92	0.36	9.62	0.71	0.89	11.4	14.9
0	100	<i>Bristol</i>	0.7without		0.94	17.1	12.1	1.10	0.76	13.3	14.1
0	100	<i>Bristol</i>	0.08without		0.75	4.23	16.6	1.39	0.91	13.2	14.7
0	100	<i>Bristol</i>	0.1without		0.67	7.34	13.9	1.55	0.98	13.1	16.7
MEAN					0.82	7.26	13.0	1.19	0.89	12.8	15.1
0	100	<i>Lipton</i>	with		0.81	1.05	13.3	0.68	0.78	16.9	17.0
0	100	<i>Lipton</i>	with		0.85	0.43	14.0	1.26	0.71	14.7	16.8
0	100	<i>Lipton</i>	with		0.77	7.24	15.8	1.42	0.74	13.6	15.7
0	100	<i>Lipton</i>	with		0.74	8.16	15.6	1.41	0.83	9.60	14.8
MEAN					0.79	4.22	14.7	1.19	0.76	13.7	16.1
0	100	<i>Lipton</i>	0.04without		0.77	0.80	13.1	0.86	0.72	15.0	14.9
0	100	<i>Lipton</i>	0.09without		0.69	2.52	13.0	1.28	0.70	14.6	15.7
0	100	<i>Lipton</i>	0.14without		0.71	8.10	15.2	1.23	0.84	11.1	15.8
0	100	<i>Lipton</i>	0.09without		0.72	6.80	10.6	1.39	0.94	13.6	14.9
MEAN					0.72	4.56	13.0	1.19	0.80	13.6	15.3
0	200	<i>Bristol</i>	with		0.81	0.54	9.34	1.71	0.88	18.5	18.0
0	200	<i>Bristol</i>	with		0.74	0.40	9.87	1.06	0.64	22.6	17.3
0	200	<i>Bristol</i>	with		0.64	7.21	11.8	1.35	0.73	12.9	16.3
0	200	<i>Bristol</i>	with		0.69	9.68	14.2	1.26	0.73	12.7	18.6
MEAN					0.72	4.46	11.3	1.35	0.75	16.7	17.5
0	200	<i>Bristol</i>	0.03without		0.66	0.25	12.9	0.92	0.61	12.2	16.9
0	200	<i>Bristol</i>	0.05without		0.90	3.18	13.2	1.45	0.53	12.2	16.3
0	200	<i>Bristol</i>	0.63without		0.71	15.8	12.4	1.49	0.76	13.6	16.8
0	200	<i>Bristol</i>	0.26without		0.65	13.7	12.6	1.38	0.79	12.2	8.07
MEAN					0.73	8.23	12.8	1.31	0.67	12.6	14.5

Table A.17: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		Glutathione		Cysteine		Glutathione	
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected
				2001/2002				2002/2003			
				μmol g ⁻¹							
0	200	<i>Lipton</i>	with	0.04	0.67	1.24	12.3	1.48	0.66	14.2	16.5
0	200	<i>Lip005</i>	with		0.59	1.09	9.24	1.22	0.68	11.6	17.7
0	200	<i>Lip007</i>	with		0.82	8.51	13.1	1.08	0.74	11.7	17.2
0	200	<i>Lip002</i>	with		0.73	4.66	15.3	1.61	0.73	13.6	17.5
MEAN					0.70	3.87	12.5	1.35	0.70	12.8	17.2
0	200	<i>Lipton</i>	0.04without		0.76	1.86	12.7	1.31	0.70	18.4	16.6
0	200	<i>Lipton</i>	1.09without		0.93	8.26	14.9	0.79	0.66	20.2	18.2
0	200	<i>Lipton</i>	0.2without		0.70	13.8	13.6	1.14	0.63	14.8	16.4
0	200	<i>Lipton</i>	0.23without		0.76	15.6	14.4	1.07	0.73	15.6	16.7
MEAN					0.79	9.87	13.9	1.08	0.68	17.3	17.0
100	100	<i>Bri009</i>	with		1.06	0.49	12.3	1.12	1.01	19.8	15.1
100	100	<i>Bri023</i>	with		1.06	12.5	13.1	1.36	1.00	12.3	16.5
100	100	<i>Bri009</i>	with		0.89	17.8	15.2	1.73	0.91	13.0	13.9
100	100	<i>Bri008</i>	with		0.87	19.3	16.2	1.68	1.18	12.5	15.4
MEAN					0.97	12.5	14.2	1.47	1.03	14.4	15.2
100	100	<i>Bristol</i>	0.03without		0.84	1.04	13.0	1.77	1.10	13.4	15.8
100	100	<i>Bristol</i>	0.05without		0.91	2.15	11.4	2.02	0.97	13.9	16.4
100	100	<i>Bristol</i>	0.22without		0.85	9.51	13.7	1.51	1.01	11.8	15.3
100	100	<i>Bristol</i>	0.53without		0.88	8.09	15.7	1.50	1.13	14.0	8.44
MEAN					0.87	5.20	13.5	1.70	1.05	13.3	14.0
100	100	<i>Lip002</i>	with		1.21	4.94	15.4	1.21	1.17	22.2	16.6
100	100	<i>Lip006</i>	with		0.97	13.9	13.4	0.84	0.81	23.4	15.6
100	100	<i>Lip001</i>	with		0.91	10.8	14.7	0.96	0.88	9.55	23.1
100	100	<i>Lip001</i>	with		0.91	17.5	16.7	1.63	0.86	11.1	7.91
MEAN					1.00	11.8	15.0	1.16	0.93	16.6	15.8
100	100	<i>Lipton</i>	0.05without		0.93	1.14	14.3	0.97	0.91	12.3	15.4
100	100	<i>Lipton</i>	0.20without		0.48	12.4	20.9	1.25	0.89	17.5	17.1
100	100	<i>Lipton</i>	0.19without		0.77	13.1	13.6	0.94	0.87	9.00	15.1
100	100	<i>Lipton</i>	0.70without		0.89	9.45	16.2	1.71	0.89	14.2	15.0
MEAN					0.77	9.02	16.2	1.22	0.89	13.2	15.6

Table A.17: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	Cysteine		Glutathione		Cysteine		Glutathione					
				infected	non-infected	infected	non-infected	infected	non-infected	infected	non-infected				
				2001/2002								2002/2003			
				$\mu\text{mol g}^{-1}$											
100	200	<i>Bristol</i>	with	1.06	0.97	17.6	13.9	1.19	0.99	25.1	17.1				
100	200	<i>Bristol</i>	with		0.95	14.8	14.4	1.13	0.89	10.9	17.0				
100	200	<i>Bristol</i>	with		0.86	15.5	13.2	1.52	0.84	12.9	17.4				
100	200	<i>Bristol</i>	with		0.85	17.2	16.0	1.85	0.94	14.9	17.3				
		MEAN			0.91	16.3	14.4	1.42	0.92	16.0	17.2				
100	200	<i>Bristol</i>	0.06without		0.81	0.71	14.2	1.14	0.92	15.8	17.9				
100	200	<i>Bristol</i>	0.03without		0.75	0.51	14.6	1.93	0.78	19.8	16.1				
100	200	<i>Bristol</i>	0.39without		0.88	15.7	13.1	1.68	0.92	15.5	7.47				
100	200	<i>Bristol</i>	0.56without		0.83	17.6	15.4	1.70	0.95	15.5	15.2				
		MEAN			0.82	8.64	14.3	1.61	0.89	16.6	14.2				
100	200	<i>Lipton</i>	with		0.84	13.3	12.6	1.16	0.89	25.3	15.6				
100	200	<i>Lipton</i>	with		1.03	7.58	13.2	1.50	0.70	12.6	18.4				
100	200	<i>Lipton</i>	with		0.81	9.31	16.3	1.61	0.80	12.4	17.5				
100	200	<i>Lipton</i>	with		0.85	15.7	12.1	1.73	1.06	13.0	9.20				
		MEAN			0.88	11.5	13.5	1.50	0.86	15.8	15.2				
100	200	<i>Lipton</i>	0.09without		0.95	4.58	16.0	0.96	0.83	14.9	17.0				
100	200	<i>Lipton</i>	0.09without		0.71	6.00	15.5	1.26	0.65	14.0	15.8				
100	200	<i>Lipton</i>	1.33without		0.88	11.9	16.1	1.42	0.86	15.3	16.5				
100	200	<i>Lipton</i>	0.87without		0.88	9.80	10.0	1.44	0.89	17.9	16.4				
		MEAN			0.86	8.08	14.4	1.27	0.81	15.5	16.4				

Table A.18: Severity of *Leptosphaeria maculans* on leaves of winter oilseed rape, Braunschweig, 2001/2002.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	<i>Leptosphaeria maculans</i> severity 1 = low; 6 = high
0	100	<i>Bristol</i>	with	4.2
0	100	<i>Bristol</i>	with	3.8
0	100	<i>Bristol</i>	with	4.6
0	100	<i>Bristol</i>	with	<u>3.8</u>
MEAN				4.1
0	100	<i>Bristol</i>	without	3.6
0	100	<i>Bristol</i>	without	3.4
0	100	<i>Bristol</i>	without	3.6
0	100	<i>Bristol</i>	without	<u>4.0</u>
MEAN				3.7
0	100	<i>Lipton</i>	with	3.2
0	100	<i>Lipton</i>	with	3.8
0	100	<i>Lipton</i>	with	4.8
0	100	<i>Lipton</i>	with	<u>4.6</u>
MEAN				4.1
0	100	<i>Lipton</i>	without	5.0
0	100	<i>Lipton</i>	without	6.0
0	100	<i>Lipton</i>	without	2.8
0	100	<i>Lipton</i>	without	<u>5.2</u>
MEAN				4.7
0	200	<i>Bristol</i>	with	3.4
0	200	<i>Bristol</i>	with	3.4
0	200	<i>Bristol</i>	with	3.2
0	200	<i>Bristol</i>	with	<u>5.0</u>
MEAN				3.8
0	200	<i>Bristol</i>	without	3.8
0	200	<i>Bristol</i>	without	3.8
0	200	<i>Bristol</i>	without	2.8
0	200	<i>Bristol</i>	without	<u>3.0</u>
MEAN				3.4
0	200	<i>Lipton</i>	with	4.6
0	200	<i>Lipton</i>	with	4.4
0	200	<i>Lipton</i>	with	5.6
0	200	<i>Lipton</i>	with	<u>5.2</u>
MEAN				5.0
0	200	<i>Lipton</i>	without	4.0
0	200	<i>Lipton</i>	without	5.2
0	200	<i>Lipton</i>	without	4.6
0	200	<i>Lipton</i>	without	<u>5.4</u>
MEAN				4.8
150	100	<i>Bristol</i>	with	3.4
150	100	<i>Bristol</i>	with	3.8
150	100	<i>Bristol</i>	with	3.0
150	100	<i>Bristol</i>	with	<u>3.6</u>
MEAN				3.5
150	100	<i>Bristol</i>	without	4.2
150	100	<i>Bristol</i>	without	4.4
150	100	<i>Bristol</i>	without	3.6
150	100	<i>Bristol</i>	without	<u>4.0</u>
MEAN				4.1

Table A.18: continued

S rate	N rate	Variety	Fungicide	<i>Leptosphaeria maculans</i> severity
kg ha ⁻¹	kg ha ⁻¹			1 = low; 6 = high
150	100	<i>Lipton</i>	with	5.2
150	100	<i>Lipton</i>	with	3.2
150	100	<i>Lipton</i>	with	3.8
150	100	<i>Lipton</i>	with	<u>5.4</u>
MEAN				4.4
150	100	<i>Lipton</i>	without	4.8
150	100	<i>Lipton</i>	without	3.8
150	100	<i>Lipton</i>	without	5.6
150	100	<i>Lipton</i>	without	<u>5.2</u>
MEAN				4.9
150	200	<i>Bristol</i>	with	3.2
150	200	<i>Bristol</i>	with	3.8
150	200	<i>Bristol</i>	with	4.2
150	200	<i>Bristol</i>	with	<u>4.6</u>
MEAN				4.0
150	200	<i>Bristol</i>	without	2.4
150	200	<i>Bristol</i>	without	3.4
150	200	<i>Bristol</i>	without	3.4
150	200	<i>Bristol</i>	without	<u>4.8</u>
MEAN				3.5
150	200	<i>Lipton</i>	with	4.8
150	200	<i>Lipton</i>	with	4.2
150	200	<i>Lipton</i>	with	5.4
150	200	<i>Lipton</i>	with	<u>5.0</u>
MEAN				4.9
150	200	<i>Lipton</i>	without	4.2
150	200	<i>Lipton</i>	without	4.0
150	200	<i>Lipton</i>	without	4.8
150	200	<i>Lipton</i>	without	<u>5.4</u>
MEAN				4.6

Table A.19: Disease assessments in winter oilseed rape crop before and after S fertilisation in spring, Aberdeen, 2001/2002.

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	4 March 2002						8 April 2002						
				S Pb	P Pb	S Lm	P Lm	S Pp	P Pp	S Pb	P Pb	S Lm	P Lm	S Pp	P Pp	
0	100	Bristol	with	0	0	0	0	0.10	20	5.90	100	0	0	0	0	0
0	100	Bristol	with	0	0	0	0	0	0	7.91	100	0	0	0	0	0
0	100	Bristol	with	0.16	30	0.06	20	0	0	3.40	70	0	0	0	0	0
0	100	Bristol09	with	—	50	0.02	0	20	0	2.55	0	80	0	—	0.21	40
MEAN					20	0.02	10	0.03	5	4.94	87.5	0	0	0	0.05	10
0	100	Bristol	without	0.70	40	0	0	0	0	5.55	100	0	0	0	0.20	20
0	100	Bristol	without	0.76	50	0	0	0	0	10.6	100	0	0	0	0	0
0	100	Bristol	without	1.00	20	0.15	20	0.05	10	26.5	100	0	0	0	0	0
0	100	Bristol	without	10.8	0	0	0	—	0	22.4	0	100	0	0	—	0
MEAN					45	0.04	5	0.01	2.5	16.3	100	0	0	0	0.05	5
0	100	Lipton	with	0	0	0	0	0.20	20	5.30	100	0	0	0	1.46	80
0	100	Lipton	with	1.65	30	0.05	10	0.02	20	10.8	100	0.06	20	0.45	50	50
0	100	Lipton	with	0.06	20	0.01	10	0	0	0	0	0	0	0	0	0
0	100	Lipton12	with	—	40	0.05	0	10	0	3.16	0	90	0	—	0.25	30
MEAN					22.5	0.03	7.5	0.06	10	4.82	72.5	0.02	5	0.54	40	40
0	100	Lipton	without	0.80	20	0.10	10	0.10	10	5.40	100	0.10	20	0	0	0
0	100	Lipton	without	0	0	0	0	0	0	14.5	100	0	0	0.20	20	20
0	100	Lipton	without	1.21	40	0.02	20	0.06	20	43.1	100	0	0	0	0	0
0	100	Lipton	without	—	50	0.05	0	10	0	0	0	0	0	0	0	0
MEAN					27.5	0.04	10	0.04	7.5	15.8	75	0.03	5	0.05	5	5
0	200	Bristol	with	0.90	30	0	0	0.10	10	2.04	100	0	0	0	0	0
0	200	Bristol	with	5.20	40	0	0	0.02	20	3.12	100	0	0	0.8	50	50
0	200	Bristol	with	0.05	10	0.05	10	0	0	0	0	0	0	0	0	0
0	200	Bristol	with	—	0	0	0	—	0	1.07	0	50	0	—	2.50	70
MEAN					20	0.01	2.5	0.03	7.5	1.56	62.5	0	0	0.83	30	30
0	200	Bristol	without	0	0	0.05	10	0	0	10.1	100	0.01	10	0.01	10	10
0	200	Bristol	without	4.65	70	0.11	30	0	0	17.0	100	0.02	20	0	0	0
0	200	Bristol	without	9.40	80	0.01	10	0	0	18.5	100	0.01	10	0	0	0
0	200	Bristol	without	—	0	0	0	—	0	8.60	0	100	0	—	0.05	10
MEAN					57.5	0.04	12.5	0	0	13.6	100	0.01	10	0.02	10	5

Table A.19: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	4 March 2002						8 April 2002					
				S Pb	P Pb	S Lm	P Lm	S Pp	P Pp	S Pb	P Pb	S Lm	P Lm	S Pp	P Pp
0	200	Lipton	with	0	0	0.70	20	0.50	30	3.31	90	0.06	20	0.20	40
0	200	Lipton	with	4.36	40	0	0	0	0	6.80	100	0.05	10	0.15	30
0	200	Lipton	with	0.20	10	0.01	10	0	0	0	0	0	0	0	0
0	200	LipD06	with	—	30	0.03	0 30	—	0	3.40	0 90	0	—	0.35	40
MEAN					20	0.19	15	0.13	7.5	3.38	70	0.03	7.5	0.18	27.5
0	200	Lipton	without	0.80	50	0	0	0.30	20	8.20	100	0	0	0.75	40
0	200	Lipton	without	0.16	30	0.06	20	0.06	20	27.4	100	0	0	0	0
0	200	Lipton	without	1.26	60	0.03	20	0.03	30	9.10	100	0	0	0	0
0	200	Lipton	without	—	40	0.17	0 40	—	0	0 0	0	0	0	—	0
MEAN					45	0.07	20	0.10	17.5	11.2	75	0	0	0.19	10
100	100	Bristol	with	0.30	20	0.25	20	0.40	40	5.21	100	0	0	0.15	20
100	100	Bristol	with	0.16	40	0	0	0.03	30	7.00	100	0	0	0.05	10
100	100	Bristol	with	0.35	20	0.01	10	0	0	6.42	100	0.01	10	0	0
100	100	Bris087	with	—	40	0.06	0 20	—	0	0 0	0	0	0	—	0
MEAN					30	0.08	12.5	0.11	18	4.66	75	0	2.5	0.05	7.5
100	100	Bristol	without	3.10	50	0.05	10	0	0	8.00	100	0	0	0	0
100	100	Bristol	without	2.65	70	0.05	10	0	0	6.70	100	0	0	0	0
100	100	Bristol	without	3.53	80	0	0	0	0	10.0	100	0	0	0	0
100	100	Bristol	without	—	0 30	0	0	—	0	6.4	0 100	0	0	—	0
MEAN					57.5	0.03	5	0	0	7.78	100	0	0	0	0
100	100	Lipton	with	0	0	0.30	20	1.70	40	8.80	90	0	0	0	0
100	100	Lipton	with	0.50	20	0.11	20	0.05	10	3.60	100	0	0	1.01	70
100	100	Lipton	with	0.10	10	0.06	20	0	0	0.90	30	0	0	0	0
100	100	Lip013	with	—	40	0.01	0 10	—	0	0.02	20	0.05	0 10	—	0
MEAN					17.5	0.12	17.5	0.44	12.5	3.33	60.0	0.01	2.5	0.25	17.5
100	100	Lipton	without	0	0	0.30	20	0	0	8.20	100	0	0	0.25	20
100	100	Lipton	without	0.20	20	0.20	10	0.02	20	13.0	100	0	0	0	0
100	100	Lipton	without	2.25	40	0.10	20	0.10	20	32.8	100	0.01	10	0	0
100	100	Lipton	without	0	0 0	0	0	—	0	0.05	0 10	0	0	—	0
MEAN					15	0.15	12.5	0.03	10	13.5	77.5	0	2.5	0	5

Table A.19: continued

S rate kg ha ⁻¹	N rate kg ha ⁻¹	Variety	Fungicide	4 March 2002					8 April 2002						
				S Pb	P Pb	S Lm	P Lm	S Pp	P Pp	S Pb	P Pb	S Lm	P Lm	S Pp	P Pp
100	200	Bristol	with	0.80	20	0.10	10	0.45	50	2.91	100	0.02	20	0.60	20
100	200	Bristol	with		10	0	0	0.03	30	8.65	100	0	0	0	0
100	200	Bristol	with		50	0.03	10	0	0	0.10	10	0	0	0	0
100	200	Bristol	with	0	—	0.00	0	—	0	6.60	0	—	0	0.35	20
MEAN					20	0.03	5	0.12	20	4.57	75	0.01	5	0.24	10
100	200	Bristol	with		20	0.15	20	0	0	16.6	100	0	0	0	0
100	200	Bristol	with		70	0	0	0	0	4.90	100	0	0	0.05	10
100	200	Bristol	with		70	0.11	30	0.01	10	15.5	100	0	0	0	0
100	200	Bristol	with	—	0	0	0	—	0	7.10	0	—	0	—	0
MEAN					42.5	0.07	12.5	0	2.5	11.0	100	0	0	0.01	2.5
100	200	Lipton	with		20	0	0	0.40	30	4.30	70	0	0	0.27	40
100	200	Lipton	with		10	0	0	0	0	7.70	90	0	0	0.46	40
100	200	Lipton	with		20	0	0	0.05	10	0	0	0	0	0	0
100	200	Lipton	with	—	20	0.01	0	—	0	4.55	100	0.05	10	0.35	30
MEAN					17.5	0	2.5	0.11	10	4.14	65	0.01	2.5	0.27	27.5
100	200	Lipton	with		50	0	0	0.20	10	6.80	100	0.05	10	0.05	10
100	200	Lipton	with		70	0.05	10	0.30	30	19.1	100	0.01	10	0	0
100	200	Lipton	with		50	0.36	30	0.01	10	20.8	100	0	0	0	0
100	200	Lipton	with	—	80	0.15	0	—	0	8.30	0	—	0	—	0
MEAN					62.5	0.14	15	0.13	12.5	13.8	100	0.02	5	0.01	2.5

note: S Pb – disease severity of *P. brassicae*; P Pb – disease incidence of *P. brassicae*; S Lm – disease severity of *L. maculans*; P Lm – disease incidence of *L. maculans*;
S Pp – disease severity of *P. parasitica*; P Pp – disease incidence of *P. parasitica*

Table A.20: Disease assessments in winter oilseed rape crop before and after S fertilisation in spring, Inverness, 2001/2002.

S rate	N rate	Variety	Fungicide	S Pb	P Pb	S Pp	P Pp
				%			
kg ha ⁻¹	kg ha ⁻¹	11 March 2002					
0	0	<i>Bristol</i>	without	3.90	90	0.20	10
0	0	<i>Lipton</i>	without	<u>3.70</u>	<u>90</u>	<u>0</u>	<u>0</u>
MEAN				3.80	90	0.10	5
100	0	<i>Bristol</i>	without	2.60	60	0	0
100	0	<i>Lipton</i>	without	<u>3.10</u>	<u>80</u>	<u>0.20</u>	<u>10</u>
MEAN				2.85	70	0.10	5
				9 April 2002			
0	100	<i>Bristol</i>	with	4.60	100	0	0
0	100	<i>Bristol</i>	with	3.90	100	0	0
0	100	<i>Bristol</i>	with	3.30	100	0	0
0	100	<i>Bristol</i>	with	<u>4.50</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				4.08	100	0	0
0	100	<i>Bristol</i>	without	10.7	100	0.05	10
0	100	<i>Bristol</i>	without	21.0	100	0	0
0	100	<i>Bristol</i>	without	6.50	100	0	0
0	100	<i>Bristol</i>	without	<u>7.80</u>	<u>100</u>	<u>0.50</u>	<u>10</u>
MEAN				11.5	100	0.14	5
0	100	<i>Lipton</i>	with	5.30	100	0	0
0	100	<i>Lipton</i>	with	5.00	100	0	0
0	100	<i>Lipton</i>	with	4.50	100	0	0
0	100	<i>Lipton</i>	with	<u>6.70</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				5.38	100	0	0
0	100	<i>Lipton</i>	without	10.0	100	0.60	30
0	100	<i>Lipton</i>	without	28.0	100	0	0
0	100	<i>Lipton</i>	without	16.3	100	0	0
0	100	<i>Lipton</i>	without	<u>24.5</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				19.7	100	0.15	7.5
0	200	<i>Bristol</i>	with	2.80	100	0	0
0	200	<i>Bristol</i>	with	4.20	100	0	0
0	200	<i>Bristol</i>	with	8.00	100	0	0
0	200	<i>Bristol</i>	with	<u>3.10</u>	<u>90</u>	<u>0</u>	<u>0</u>
MEAN				4.53	97.5	0	0
0	200	<i>Bristol</i>	without	9.20	100	0.10	20
0	200	<i>Bristol</i>	without	13.4	100	0	0
0	200	<i>Bristol</i>	without	12.0	100	0	0
0	200	<i>Bristol</i>	without	<u>10.6</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				11.3	100	0	5
0	200	<i>Lipton</i>	with	5.90	100	0	0
0	200	<i>Lipton</i>	with	6.40	100	0	0
0	200	<i>Lipton</i>	with	3.00	100	0	0
0	200	<i>Lipton</i>	with	<u>5.90</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				5.30	100	0	0
0	200	<i>Lipton</i>	without	11.3	100	0	0
0	200	<i>Lipton</i>	without	11.2	100	0	0
0	200	<i>Lipton</i>	without	13.1	100	0	0
0	200	<i>Lipton</i>	without	<u>13.7</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				12.3	100	0	0

Table A.20: continued

S rate	N rate	Variety	Fungicide	S Pb	P Pb	S Pp	P Pp
				%			
kg ha ⁻¹		9 April 2002					
100	100	<i>Bristol</i>	with	4.20	100	0	0
100	100	<i>Bristol</i>	with	3.50	90	0	0
100	100	<i>Bristol</i>	with	3.00	100	0	0
100	100	<i>Bristol</i>	with	<u>6.50</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				4.30	97.5	0	0
100	100	<i>Bristol</i>	without	7.90	100	0	0
100	100	<i>Bristol</i>	without	13.2	100	0	0
100	100	<i>Bristol</i>	without	15.3	100	0	0
100	100	<i>Bristol</i>	without	<u>5.40</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				10.5	100	0	0
100	100	<i>Lipton</i>	with	4.90	100	0	0
100	100	<i>Lipton</i>	with	5.40	100	0	0
100	100	<i>Lipton</i>	with	5.10	100	0	0
100	100	<i>Lipton</i>	with	<u>2.30</u>	<u>80</u>	<u>0</u>	<u>0</u>
MEAN				4.43	95	0	0
100	100	<i>Lipton</i>	without	10.0	100	0	0
100	100	<i>Lipton</i>	without	13.1	100	0	0
100	100	<i>Lipton</i>	without	10.8	100	0	0
100	100	<i>Lipton</i>	without	<u>11.3</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				11.3	100	0	0
100	200	<i>Bristol</i>	with	4.50	100	0	0
100	200	<i>Bristol</i>	with	1.80	100	0	0
100	200	<i>Bristol</i>	with	4.20	100	0	0
100	200	<i>Bristol</i>	with	<u>2.80</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				3.33	100	0	0
100	200	<i>Bristol</i>	without	9.70	100	0	0
100	200	<i>Bristol</i>	without	8.00	100	0.60	20
100	200	<i>Bristol</i>	without	9.10	100	0	0
100	200	<i>Bristol</i>	without	<u>16.3</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				10.8	100	0.15	5
100	200	<i>Lipton</i>	with	3.30	100	0	0
100	200	<i>Lipton</i>	with	2.50	80	0	0
100	200	<i>Lipton</i>	with	4.00	100	0	0
100	200	<i>Lipton</i>	with	<u>5.90</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				3.93	95	0	0
100	200	<i>Lipton</i>	without	11.7	100	0	0
100	200	<i>Lipton</i>	without	15.5	100	0	0
100	200	<i>Lipton</i>	without	12.4	100	0	0
100	200	<i>Lipton</i>	without	<u>9.10</u>	<u>100</u>	<u>0</u>	<u>0</u>
MEAN				12.2	100	0	0

note: S Pb – disease severity of *P. brassicae*; P Pb – disease incidence of *P. brassicae*; S Pp – disease severity of *P. parasitica*; P Pp – disease incidence of *P. parasitica*

Table A.21: Disease assessments in winter oilseed rape crop before and after S fertilisation in spring, Aberdeen, 2002/2003.

S rate	N rate	Variety	Fungicide	S Pb	P Pb
				%	
kg ha ⁻¹	kg ha ⁻¹			10 March	
0	0	<i>Bristol</i>	without	5.8	100
0	0	<i>Lipton</i>	without	<u>7.3</u>	<u>100</u>
MEAN				6.6	100
100	0	<i>Bristol</i>	without	7.3	100
100	0	<i>Lipton</i>	without	<u>7.4</u>	<u>100</u>
MEAN				7.4	100
24 March					
0	0	<i>Bristol</i>	without	4.5	60
0	0	<i>Lipton</i>	without	<u>1.1</u>	<u>50</u>
MEAN				2.8	55
100	0	<i>Bristol</i>	without	1.3	70
100	0	<i>Lipton</i>	without	<u>1.0</u>	<u>50</u>
MEAN				1.2	60
28 April					
0	100	<i>Bristol</i>	with	6.4	100
0	100	<i>Bristol</i>	with	9.5	100
0	100	<i>Bristol</i>	with	10.0	100
0	100	<i>Bristol</i>	with	<u>7.3</u>	<u>100</u>
MEAN				8.3	100
0	100	<i>Bristol</i>	without	4.7	90
0	100	<i>Bristol</i>	without	3.3	100
0	100	<i>Bristol</i>	without	3.4	80
0	100	<i>Bristol</i>	without	<u>4.7</u>	<u>90</u>
MEAN				4.0	90
0	100	<i>Lipton</i>	with	10.1	100
0	100	<i>Lipton</i>	with	16.7	100
0	100	<i>Lipton</i>	with	8.2	100
0	100	<i>Lipton</i>	with	<u>9.7</u>	<u>100</u>
MEAN				11.2	100
0	100	<i>Lipton</i>	without	4.7	80
0	100	<i>Lipton</i>	without	3.4	70
0	100	<i>Lipton</i>	without	1.9	90
0	100	<i>Lipton</i>	without	<u>2.5</u>	<u>90</u>
MEAN				3.1	82.5
0	200	<i>Bristol</i>	with	18.9	100
0	200	<i>Bristol</i>	with	20.2	100
0	200	<i>Bristol</i>	with	5.0	100
0	200	<i>Bristol</i>	with	<u>3.5</u>	<u>90</u>
MEAN				11.9	97.5
0	200	<i>Bristol</i>	without	7.0	100
0	200	<i>Bristol</i>	without	4.1	90
0	200	<i>Bristol</i>	without	7.9	90
0	200	<i>Bristol</i>	without	<u>3.1</u>	<u>90</u>
MEAN				5.5	92.5

Table A.21: continued

S rate	N rate	Variety	Fungicide	S Pb	P Pb
				%	
kg ha ⁻¹	kg ha ⁻¹			28 April	
0	200	<i>Lipton</i>	with	8.2	100
0	200	<i>Lipton</i>	with	10.7	100
0	200	<i>Lipton</i>	with	12.1	100
0	200	<i>Lipton</i>	with	<u>11.1</u>	<u>100</u>
MEAN				10.5	100
0	200	<i>Lipton</i>	without	3.0	90
0	200	<i>Lipton</i>	without	3.4	80
0	200	<i>Lipton</i>	without	5.0	90
0	200	<i>Lipton</i>	without	<u>5.8</u>	<u>80</u>
MEAN				4.3	85
100	100	<i>Bristol</i>	with	15.4	100
100	100	<i>Bristol</i>	with	13.2	100
100	100	<i>Bristol</i>	with	3.2	100
100	100	<i>Bristol</i>	with	<u>10.7</u>	<u>100</u>
MEAN				10.6	100
100	100	<i>Bristol</i>	without	2.8	100
100	100	<i>Bristol</i>	without	3.4	90
100	100	<i>Bristol</i>	without	3.7	90
100	100	<i>Bristol</i>	without	<u>5.6</u>	<u>90</u>
MEAN				3.9	92.5
100	100	<i>Lipton</i>	with	12.5	100
100	100	<i>Lipton</i>	with	12.9	100
100	100	<i>Lipton</i>	with	5.8	100
100	100	<i>Lipton</i>	with	<u>14.7</u>	<u>100</u>
MEAN				11.5	100
100	100	<i>Lipton</i>	without	2.8	80
100	100	<i>Lipton</i>	without	3.5	80
100	100	<i>Lipton</i>	without	3.5	90
100	100	<i>Lipton</i>	without	<u>4.8</u>	<u>90</u>
MEAN				3.7	85
100	200	<i>Bristol</i>	with	8.0	100
100	200	<i>Bristol</i>	with	7.4	100
100	200	<i>Bristol</i>	with	6.5	100
100	200	<i>Bristol</i>	with	<u>9.9</u>	<u>100</u>
MEAN				8.0	100
100	200	<i>Bristol</i>	without	2.9	80
100	200	<i>Bristol</i>	without	8.9	90
100	200	<i>Bristol</i>	without	4.4	70
100	200	<i>Bristol</i>	without	<u>6.5</u>	<u>100</u>
MEAN				5.7	85
100	200	<i>Lipton</i>	with	23.2	100
100	200	<i>Lipton</i>	with	11.1	100
100	200	<i>Lipton</i>	with	5.7	90
100	200	<i>Lipton</i>	with	<u>17.7</u>	<u>100</u>
MEAN				14.4	97.5
100	200	<i>Lipton</i>	without	5.2	100
100	200	<i>Lipton</i>	without	4.4	70
100	200	<i>Lipton</i>	without	2.0	80
100	200	<i>Lipton</i>	without	<u>6.8</u>	<u>90</u>
MEAN				4.6	85

note: S Pb – disease severity of *P. brassicae*; P Pb – disease incidence of *P. brassicae*

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