Control of fungal diseases in winter wheat

Evaluation of long-term field research in southern Sweden

Lars Wiik
Faculty of Landscape Planning, Horticulture and Agricultural Sciences
Department of Plant Protection Biology
Alnarp

Doctoral Thesis
Swedish University of Agricultural Sciences
Alnarp 2009

Acta Universitatis agriculturae Sueciae
2009:97

Cover: Severe attacks of septoria tritici blotch on older leaves with pycnidia in the spring before stem elongation (photo: Peder Waern)

ISSN 1652-6880
ISBN 978-91-576-7444-9

Print: SLU Service/Repro, Alnarp 2009

Control of fungal diseases in winter wheat – Evaluation of long-term field research in southern Sweden

Abstract

The relationships between plant diseases, winter wheat characteristics, air temperature and precipitation, site factors and agricultural practices were investigated. Regression analyses revealed that control of LBDs (Leaf Blotch Diseases, including septoria tritici blotch, stagonospora nodorum blotch and tan spot) explained 74% of the yield increase achieved by fungicide treatment at GS 45-61, followed by powdery mildew (20%), brown rust (5%) and yellow rust (1%). Yield of both untreated and fungicide-treated plots increased from 6000 to 12000 kg ha^{-1} over the period 1983-2005. Single eyespot treatment improved mean yield by ~320 kg ha^{-1} yr^{-1} during the period 1977-2002, mainly due to occasional years with severe eyespot. A fungicide treatment at GS 45-61 increased mean yield by 10.3% or 810 kg ha^{-1} yr^{-1} (9.9% or 660 kg ha^{-1} yr^{-1} for 1983-1994 and 10.7% or 970 kg ha^{-1} yr^{-1} for 1995-2005) due to increased TGW and grain numbers, especially in high yielding stands. Air temperature and precipitation as monthly means explained more than 50% of the variation between years regarding yield increase, TGW, LBDs, brown rust, yellow rust and eyespot, but less than 50% of the variation in yield and powdery mildew. Precipitation in May was the factor most consistently related to LBD disease intensity, and adding another two weather factors further improved the degree of explanation. Weather factors in the preceding growing season influenced growth stage, powdery mildew and brown rust. Mild winters and springs favoured the biotrophs, *i.e.* powdery mildew, brown rust and yellow rust. The mean net return from fungicide use was negative in 10 years and less than 50% of the entries were profitable to treat in 11 years. Fungicide use was in fact more profitable (mean net return 21 compared with 3 € ha^{-1}) during the latter part of the period (1995-2007) than in the earlier part (1983-1994). The role of site factors and agricultural factors is complex but some factors, such as pre-crop and dose of nitrogen, can probably be used in plant disease warning and prediction models. Wheat as pre-crop to wheat gave 1.6 tons ha^{-1} lower yield than rape as pre-crop. The results confirm the potential and limits of fungicides and the need for supervised control strategies that include factors affecting disease, yield, interactions and overall profitability.

Keywords: yield loss, Septoria tritici, weather, economics, disease prediction, IPM.

Author's address: Lars Wiik, SLU, Department of Plant Protection Biology,
P.O. Box 102, SE-230 53 Alnarp, Sweden
E-mail: Lars.Wiik@ltj.slu.se

Contents

List of Publications **7**

1 Introduction **9**

2 Background **11**

2.1 Environment 11

2.2 Winter wheat 12

2.2.1 A global staple crop 12

2.2.2 An important crop in Sweden 13

2.2.3 Modern plant breeding 15

2.2.4 Yields in Sweden in recent times 16

2.2.5 Plant breeding drawbacks 18

2.3 Essentials of fungal diseases 19

2.3.1 Categorisation and identification 19

2.3.2 Crop loss assessment 21

2.3.3 Disease cycles and epidemiology 23

2.3.4 Variability and adaptation 24

2.4 Control 25

2.4.1 Fungicides 25

2.4.2 Host plant resistance 28

2.4.3 Cultural methods 37

2.5 Fungal diseases in focus 40

2.5.1 LBDs with the focus on septoria trititci blotch 40

2.5.2 Powdery mildew and the rusts 47

2.5.3 Eyespot 56

3 Aims and objectives **61**

4 Materials and methods **63**

4.1 Field experiments (Papers I-IV) 63

4.2 Surveys (Paper II) 64

4.3 Meteorological data (Paper II) 65

4.4 Economics (Paper III) 65

4.5 Site factors and agricultural practices (Paper IV) 65

4.6 Statistics 65

5 Results and discussion **66**

5.1 Grain yield quantity 66
5.2 Grain yield quality 68
5.3 Plant diseases and their importance 70
5.4 Effect of fungicides 72
5.5 Weather, plant diseases and yield increase 73
5.6 Economics 75
5.7 Site factors and agricultural practices 75

6 Conclusions and future implementations 78
6.1 Overall conclusions 81
6.1.1 Main conclusions 81
6.1.2 Detailed conclusions 81
6.2 Recommendations 82

References 83

Acknowledgements 107

List of Publications

This thesis is based on the work contained in the following papers, referred to by Roman numerals in the text:

I Wiik, L. 2009. Yield and disease control in winter wheat in southern Sweden during 1977-2005. *Crop Protection* 28 (1), 82-89.

II Wiik, L. & Ewaldz, T. 2009. Impact of temperature and precipitation on yield and plant diseases of winter wheat in southern Sweden 1983-2007. *Crop Protection* 28 (11), 952-962.

III Wiik, L. & Rosenqvist H. 2010. The economics of fungicide use in winter wheat in southern Sweden. *Crop Protection* 29 (1), 11-19.

IV Wiik, L. & Englund, J.-E. 2009. Influence of site factors and agricultural practices on yield and plant diseases of winter wheat in southern Sweden. (Manuscript).

Papers I-III are reproduced with the permission of the publishers.

The contribution of Lars Wiik to the papers included in this thesis was as follows:

I 95%

II 90%

III 85%

IV 75%

1 Introduction

This thesis takes its starting point in biology and chemistry extended to ecology and applied agricultural sciences, principally plant protection biology, supported by statistics and economics. In plant protection biology the focus is on the plant/crop, the associated pests and diseases and the control of these pests and diseases to avoid economic damage. Economic damage is avoided by several measures, the most important in modern agriculture being plant breeding resulting in resistant varieties and chemistry resulting in pesticides. In science the challenge is to explain cause and effect in nature and to use the knowledge to understand and predict coming events. Science is essential in plant protection biology as we have to explain and learn more about whether, why, where, when and how pests and diseases constrain the quantitative and qualitative yield of plants. We can predict coming epiphytotics and often control them by increased knowledge of preventative and acute methods, *e.g.* choosing the right variety and applying pesticides at the best time during the growing season.

The agroecosystems of today, which are characterised by higher yields and less diversity than agroecosystems in the past, require inputs of production resources such as high-yielding varieties, fertilizers and pesticides to fulfil the aim of high production. However, concerns about natural resources and our environment call into question the use of perceived hazardous means of production, such as use of pesticides and fertilizers. The rebirth of integrated pest management (IPM) in the EU is a sign of the current unease about the excessive dependence of modern agriculture on pesticides.

In spite of the usual cultivation of one crop at a time agricultural ecosystems are complex, *e.g.* as illustrated in the epidemiology[1] of plant

---

[1] Epidemiology was defined as the behaviour of disease in populations by van der Planck (1963).

disease, or the measured, compared and interacting influence of plant host, pathogen and environment on disease development and spread. The complexity in epidemiology has been illustrated by Zadoks & Schein (1979) by the disease tetrahedron, showing the interactions of host plant, pathogen and environment and the various effects of man. Agrios (2005) takes the concept of the disease tetrahedron or disease pyramid further and considers time to be the fourth element and human interventions a distinct fifth element. Even if one disease or pest often dominates in a field, a crop usually suffers from more than one biotic constraint, and in addition several abiotic constraints affect the crop.

Long-term results from field trials in southern Sweden (Scania) were evaluated in the present thesis in order to examine the impact of different diseases on winter wheat yield, as well as some abiotic factors affecting disease development and yield. Long-term results give us an opportunity to follow changes, *e.g.* changes in yield and disease intensity observed in relation to the time of the study, in this thesis about three decades. Furthermore, evaluation of long-term results gives us the opportunity to identify important components in IPM strategies for plant health management and sustainable agriculture.

2 Background

2.1 Environment

In Sweden four seasons – spring, summer, autumn and winter – can be distinguished during the year, mostly due to differences in temperature. Winter can be defined as the months with mean temperature less than 0°C and summer as the months with mean temperature above 10°C, but another definition is that December, January and February are the winter months, March, April and May are spring, June, July and August summer and September, October and November autumn. Changes in weather conditions affect the crop in different ways all through the growing season, from seed to end product. The impact of meteorological factors on both the crop and plant disease has been shown for several pathosystems (Rotem, 1978; Zadoks & Schein, 1979; Campbell & Madden, 1990). The rice blast fungus is dependent on three factors to cause an epiphytotic: the abundance of conidia, the infection process and host resistance, all influenced by the weather (Suzuki, 1975). Late blight on potato (Bourke & Lamb, 1993) is dependent on high relative humidity, free water on the leaves, temperature and wind. Soybean rust is dependent on the environmental variation during establishment, dispersal and establishment (Yang, 2006). Humidity/moisture and temperature are the most important physical properties when fungal pathogens are considered, but many meteorological properties are inter-correlated. Wind and rain are important for the dissemination or spread of pathogens. Weather is of paramount importance for crop growth and the relationships between the environment, the crop and the pathogens are obvious. The environment has a large effect on both the quality and yield of grain (Fajersson, 1961; Svensson, 1981; Johansson & Svensson, 1998; Jiang *et al.*, 2003). The impact of meteorological factors on winter wheat growth and its diseases has been the main focus in many investigations (Coakley, 1988; Johnsson, 1992a, 1992b; Royle *et al.*, 1993; Wiik, 1993; Khoury & Kranz, 1994; Verreet, 1995; Gladders *et al.*, 2001; Pietravalle *et al.*, 2003; Gladders *et al.*, 2007; Papastamati & van den Bosch, 2007; Shaw *et al.*, 2008; Te Beest *et al.*, 2008). The environment of a crop and its fungal pathogens is dependent on more than meteorological factors. Edaphic conditions such as soil physics and soil chemistry also affect both the crop and its diseases. Agricultural practices affect the environment in many ways, *e.g.* ploughing or no-till, fertilising and application of pesticides (Shipton, 1977; Cowling, 1978; Lévesque & Rahe, 1992; Bockus & Shroyer, 1998; Rodgers-Gray & Shaw, 2000; Simón *et al.*, 2003; Bakker *et al.*, 2005).

2.2 Winter wheat

2.2.1 A global staple crop

The winter bread wheat produced in agriculture today is a high-yielding hexaploid grass of the family *Poaceae* and is one of the main staple foods world-wide. Wheat has accompanied man during 10 000 years and evolved to its present position by continuous selection and plant breeding. Winter wheat yields have so far kept in pace with the growth of the human population. Advances have been made in agriculture step by step, with occasional major steps resulting in expansion and yield enhancement, *e.g.* fortunate crossings, from animal to tractor power, from manure/guano to nitrate deposits in Chile and further to nitrogen fertilizer production by the Haber-Bosch process, from tall, low-yielding varieties to the green revolution with short, high-yielding varieties. Wheat is grown around the globe, annually on a total of about 200 million hectares during recent years, and possesses a large capacity for adaptation (Tigerstedt, 1997). Winter wheat is still occupying the largest acreage but was overtaken by both maize and rice in terms of amounts produced during the late 1990s (The Economist, 2005). In EU-27, almost 150 million tons of wheat were produced in 2008 and world-wide at least four times more. Borlaug (2007) predicts demand for cereals to grow by 50% over the next 20 years, a challenge that will require advances in traditional science and new areas such as biotechnology. Efforts to increase yields made by the world-wide, influential International Maize and Wheat Improvement Center (CIMMYT) have so far been very successful, with an annual increase in wheat yield potential of 0.9% between 1970 and 1995 (Ortiz *et al.*, 2007).

According to Bray *et al.* (2000), the maximum potential yield of winter wheat grown under ideal conditions is 14.5 tons ha^{-1} but the actual global average yield is only 1.88 tons ha^{-1}. Those authors found losses due to biotic stress (diseases, insects and pests) to amount to an average 5% of maximum potential yield and losses due to abiotic stress (environmental factors such as drought, salinity, flooding, low and high temperature, *etc.*) to amount to 82%. Accordingly, varieties with tolerance or resistance to abiotic stress will be of major importance in improving yield.

Oerke *et al.* (1994) cite a yield potential of 18 tons ha^{-1} of modern varieties under temperate climatic conditions and higher estimated annual losses due to biotic constraints than those reported by Bray *et al.* (2000). During the period 1988-1990, Oerke *et al.* (1994) found the overall actual global losses due to diseases, animal pests and weeds to be 12.4, 9.3 and 12.3%, respectively, and corresponding losses without crop protection or potential losses to be 16.7, 11.3 and 23.9%, respectively. Overall actual European losses due to diseases, animal pests and weeds were 9, 7 and 9%, respectively, and corresponding potential losses 20, 12 and 21%, respectively, which are considerably higher than the figures given by Bray *et al.* (2000). Several authors point out the utmost importance of disease control (Klinkowski, 1970; Strange & Scott, 2005).

2.2.2 An important crop in Sweden

In Sweden, the forerunners diploid einkorn (*Triticum monococcum*), tetraploid emmer (*T. turgidum* ssp. *dicoccum*) and some hexaploid spelt wheat (*T. spelta*) have been used since the Neolithic Age 5 500 years ago, followed by common wheat (*T. aestivum*) from the 1700s onwards (Welinder *et al.*, 1998). Wheat cultivation in northern Sweden is not advisable due to the short growing season. In Central Sweden (especially in the counties of Västra Götaland, Östergötland, Uppsala, Södermanland, Västmanland and Örebro) and southern Sweden, winter wheat is usually cultivated successfully but varieties with cold hardiness and winter survival ability are important (Larsson, 1986; Svensson, 1997). The Swedish contribution to wheat production in EU-27 in 2008 was about 1.5% and the Swedish acreage is about 0.15% of the global total. Yields of winter wheat improved markedly in Sweden during the 1900s, especially from the 1960s (Figure 1).

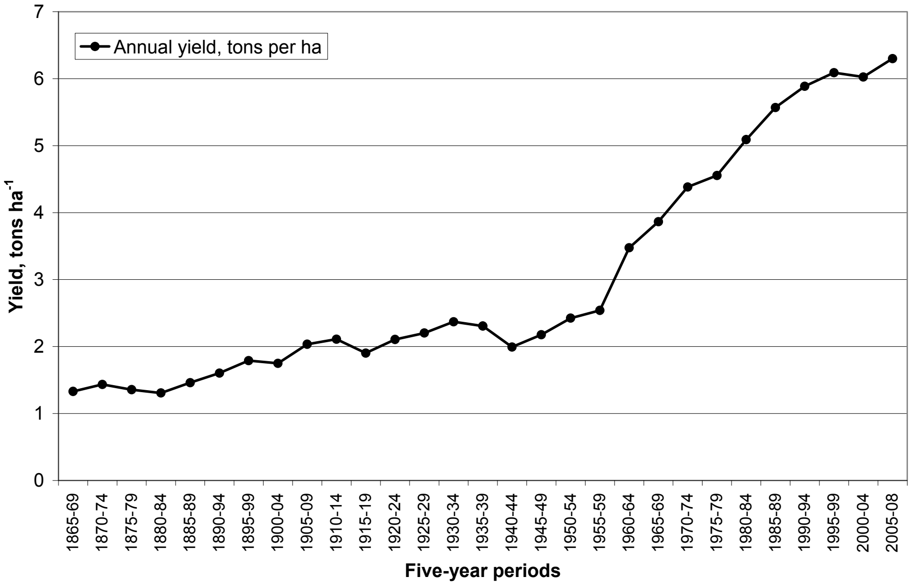

Figure 1. Mean winter wheat yields (tons ha^{-1}) in Sweden in five-year periods from 1865 to 2008 (2005-08 only four years).

During 1865-1884 (the first four five-year periods of records), mean yields of winter wheat did not exceed 1.5 tons ha^{-1}. In the following six five-year periods, 1885 until the start of the Great War in 1914, mean yields continuously increased to approximately 2 tons ha^{-1}. During the interwar period yield increased steadily, to nearly 2.5 tons ha^{-1}. Since the late 1950s until today, however, the change in yield has been remarkable, an annual mean increase of 74 kg ha^{-1} compared with 16 kg ha^{-1} during the pre-World War II period (Figure 2). These figures are similar to the mean annual grain increase of 28 kg ha^{-1} estimated by Mac Key (1993) for the period 1866-1990, similar to results from France and the United Kingdom (Austin, 1999; Brancourt-Hulmel *et al.*, 2003). Furthermore, Mac Key (1993) estimated the mean annual increase attributable to advances in plant breeding in winter wheat to be 0.55% during the period 1900-1990, *i.e.* about half the total increase, illustrating the common expression of the gene-environment interaction to be about 50/50. Yields decreased during both World Wars, *i.e.* in the five-year periods 1915-1919 and 1940-1944, most probably due to lack of fuel, fertilizers and other imports. The remarkable increase in yields since the late 1950s is associated with the increased use of fertilizers (NPK) on arable land in Sweden (Figure 3) and winter wheat breeding that started in Sweden around 1900.

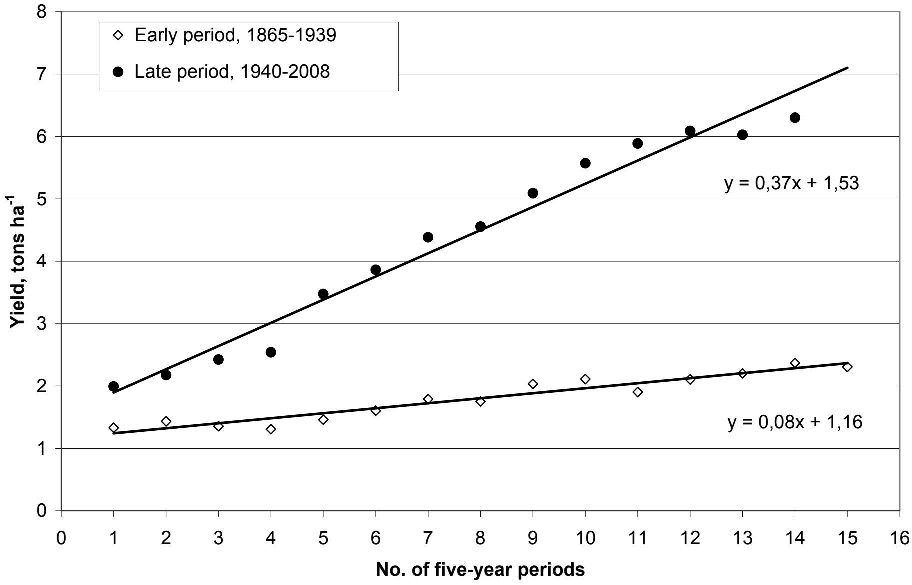

Figure 2. Mean winter wheat yields (tons ha^{-1}) in Sweden in five-year periods during two periods, an early period 1865-1939 and a late period 1940-2008.

2.2.3 Modern plant breeding

Efficient breeding and pedigree breeding of wheat in general started back in the nineteenth century, based on old landrace populations, foreign cultivars and lines such as English Squarehead wheat with at that time high yield potential and a stiffer straw (Persson *et al.*, 1986; Svensson, 1997). During the first part of the twentieth century crossings between old Swedish landraces with winter hardiness and good kernel quality and Squarehead wheat with its higher yield and stiffer straw resulted in cultivars such as Extra Squarehead II (1909), Pansar I, II and III (1915, 1919 and 1923), Sol I and II (1911 and 1916), Standard I and II (1921 and 1936) and Saxo (1929) (Nordgen, 2009).

Successful Swedish wheat breeding continued, including use of foreign traits with desirable qualities, resulting in cultivars such as Eroica (1943), Starke (1959) and Starke II (1968), followed by Holme (1972), Solid (1973), Folke (1981), Kosack (1984), Sleipner (1988), Tjelvar (1988), Rental (1993) and Stava (1995) (Nordgen, 2009). However during the past 50-60 years, significantly increased use of fertilizers, varieties with better straw strength and the use of pesticides have also contributed to the higher yields (Figure 3).

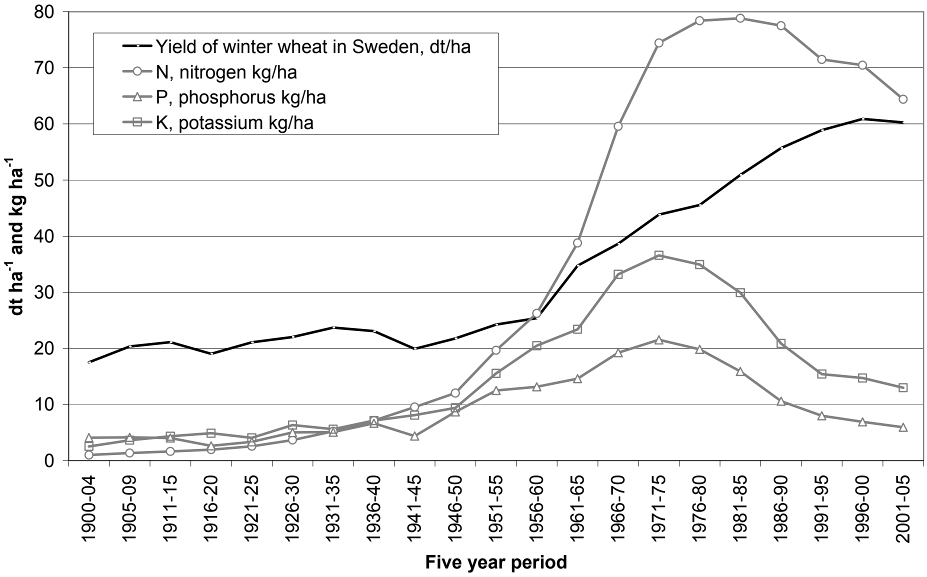

Figure 3. Yield of winter wheat (dt ha^{-1}), and use of nitrogen (kg ha^{-1}), phosphorus (kg ha^{-1}) and potassium (kg ha^{-1}) on the total arable land in Sweden during five year periods 1900 to 2005. Compiled from SJV 2009.

As an example, the mean use of nitrogen fertilizer over all crops increased from less than 10 kg ha^{-1} to more than 80 kg ha^{-1} during the period 1940-1990 (Morell, 2001). During the 1990s, high-yielding and early maturing new continental varieties were introduced in Sweden. These foreign varieties contributed to the increase in mean yields in southern Sweden shown by the results from variety field trials. The foreign varieties Ritmo (Cebeco-Zaden B.V., Vlijmen, The Netherlands), Kris (PBIS, Germany) and Marshal (Zeneca Seeds, Norfolk, England) yielded 13, 13 and 18%, respectively more than the popular Swedish variety Kosack in fungicide-treated plots in southern Sweden during 1998-2002 (Larsson *et al.*, 2003). These varieties were not as high-yielding in Central Sweden, *e.g.* Ritmo actually gave lower yields than Kosack in Central Sweden. Examples of factors limiting yield during this latter period include limited crop rotations, diminishing fungicide efficacy and single years of severe attacks of eyespot, stem-base diseases and aphids (Sigvald, 1984; Olofsson, 1993; Larsson, 2005; Bryson *et al.*, 2006).

2.2.4 Yields in Sweden in recent times

During the ten five-year periods between 1958 and 2007, yield was 0.78 ton ha^{-1} higher in southern Sweden than in Sweden as a whole (Figure 4).

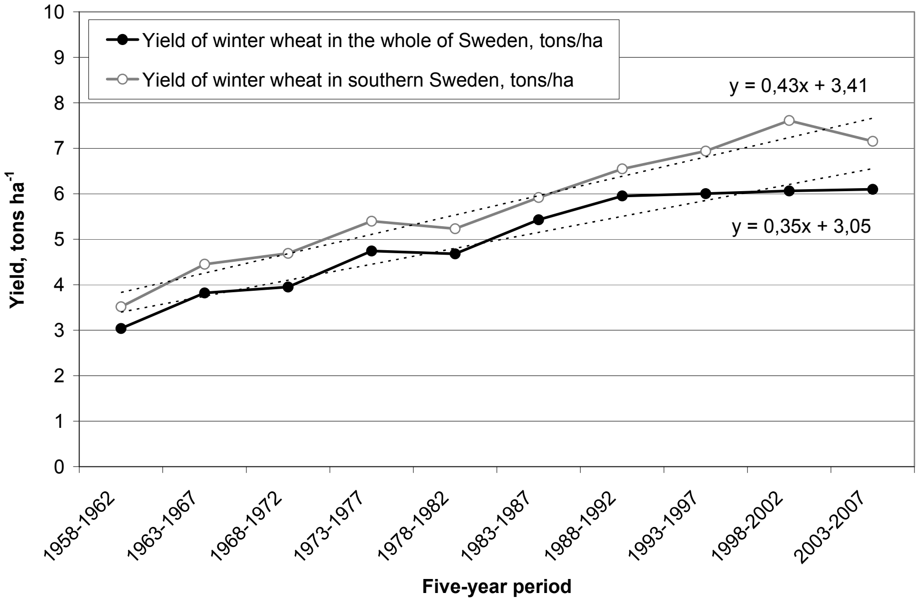

Figure 4. Mean winter wheat yields (tons ha^{-1}) in the whole of Sweden and in southern Sweden (Scania) in five-year periods from 1958 to 2007.

A contributing factor to the lower yields in Sweden as a whole was the larger bread wheat than feed wheat acreage in Central Sweden than in southern Sweden (80/20 bread/fodder wheat acreage in Central Sweden compared with 50/50 in the south) (Hans Thorell, Lantmännen, pers. comm. October 2009). From the beginning of the 1990s yields levelled out in Sweden as a whole but in southern Sweden the increase in yields continued for another ten years. The explanation for this difference may be that continental varieties are more adapted to the climate in southern Sweden than in Central Sweden, a slower introduction of high-yielding varieties in Central Sweden, and maybe also a faster learning from the progress in neighbouring southern countries.

It is a well-known fact that yields in field trials are higher than those obtained in practical farming, *e.g.* as in fungicide field trials carried out 1983-2007 in southern Sweden (Figure 5). This can probably be explained by better soils, cultivars and agricultural practices in the field trials than in wheat crops in general. However, the slope of the yield curve in national statistics is less steep than the slope of the curves in the field trials, which indicates a faster increase in yields achieved in field trials. This might be explained by delayed transfer of new knowledge to practice, as found in England and

Wales, where it took 10 years to change the timing of fungicide application (Cook *et al.*, 1999; Hardwick *et al.*, 2001).

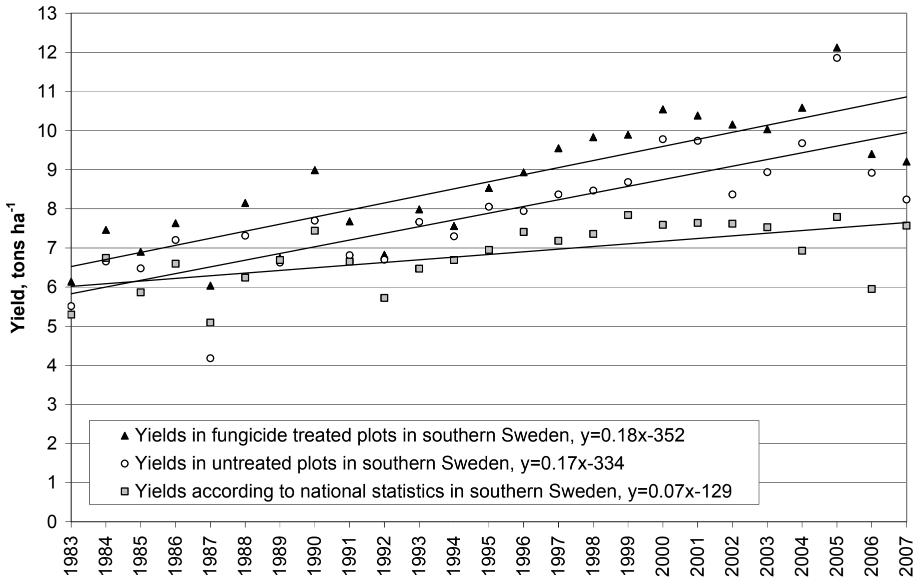

Figure 5. A comparison of yield development in fungicide field trials with untreated and with fungicide treated plots and the yields by the national agricultural statistics 1983-2007.

In a field trial in southern Sweden during 2000, the highest mean yield was 15.38 tons ha^{-1} at 85% dry matter in four fungicide-treated plots with the fodder wheat cultivar Marshal from Zeneca Seeds, Norfolk, England (Ljungars, 2001). Marshal was at that time the highest yielding winter wheat variety tested in southern Sweden (Larsson *et al.*, 2001), and the maximum yield recorded was higher than the record yield of 14.5 tons ha^{-1} reported by Bray *et al.* (2000). Plant breeding and agricultural practices will most likely contribute to even higher winter wheat yields in the future, as suggested by Oerke *et al.* (1994). Record yields of more than 20 tons ha^{-1} and mean yields of about 15 tons ha^{-1} from fields with good agricultural soils and favourable meteorological conditions are probably not far away.

2.2.5 Plant breeding drawbacks

Plant breeding is a difficult task as a number of factors have to be considered. Both the quantity and quality of yield are important goals, not at least quantity or high agronomic yield, to meet the demands of a hungry world. Quality of wheat grain includes factors affecting milling, baking, processing qualities and nutritional effects, such as different aspects of seed health, kernel protein (gluten), kernel starch (amylopectin and amylose) and fibre, kernel hardness, kernel density, kernel size and hectolitre weight, endosperm proportion, separation ability between endosperm and bran, vitamin content and content of minerals such as calcium, magnesium, potassium, phosphorus, sodium and iron, essential and basic amino acids, antioxidant phytochemicals and bioactive compounds. Concerns have been raised about loss of quality during breeding focusing on quantity (Morris & Sands, 2006). In terms of minerals in the kernel, the concentrations of zinc, iron, copper and magnesium have decreased since the 1960s in wheat samples from the Broadbalk Wheat Experiment at Rothamsted, UK. This has coincided with the introduction of the semi-dwarf high-yielding varieties (Fan *et al.*, 2008). A corresponding decrease in minerals and other traits has been reported elsewhere (Welch & Graham, 1999; Garvin *et al.*, 2006). Zhao *et al.* (2009) found higher selenium concentrations in spelt, einkorn and emmer than in bread and durum wheats. Specific breeding programmes have been proposed to maintain acceptable quality (Morris & Sands, 2006). Other drawbacks are genetic vulnerability – the opposite of diversity – due to the plant genetic uniformity created when a variety is introduced on a considerable acreage and loss of resistance to a range of pests and diseases (Johnson, 1984; Pring & Lonsdale, 1989; Browning, 1998; Reif *et al.*, 2005).

2.3 Essentials of fungal diseases

2.3.1 Categorisation and identification

Fungal diseases are one of several biotic constraints or restrictions to winter wheat yields. The book Nordic Names of Plant Diseases and Pathogens (Gjaerum *et al.*, 1985) lists 34 different fungal diseases on *T. aestivum* L., but on a world-wide basis many more are to be found (Wiese, 1977). Serious fungal diseases include rusts and smuts, including bunts. Black stem rust (*Puccinia graminis* f.sp. *graminis*), brown rust (leaf rust) (*P. triticina*) and yellow rust (stripe rust) (*P. striiformis* f.sp. *tritici*) are major, colourful and occasionally devastating rust diseases on wheat. The smuts are easily recognised by the replacement of the grains by spore masses, while the bunts, both common bunt (*Tilletia tritici*, syn. *T. caries*) and dwarf bunt (*Tilletia controversa*, syn. *T. contraversa*), are characterised by an awful smell.

There are a few different principles of fungal disease categorisation:

- species related to each other like the rusts and like the smuts,

- whether they are biotrophic, defined as pathogens that obtain nutrients from living host tissue, or necrotrophic, often toxin-producing fungi that obtain their energy from dead tissue,
- the part of the plant that is colonised or attacked,
- where in the plant they are most frequently found,
- how they are promoted and spread
- other categorisation principles, such as preharvest and postharvest diseases.

The seed, root, stem-base, stem, foliage and ear are common parts of the plant occupied or invaded by fungal pathogens, but a specific fungus is usually not restricted to only one part and might for example be a seed, foliar and ear disease. Common seedborne pathogens are the above mentioned smuts, several *Fusarium* species including mycotoxin producers, *Pyrenophora tritici-repentis*, *Microdochium nivale* and *Phaeosphaeria nodorum* (*anamorph Stagonospora nodorum*). Root and stem-base diseases include take-all (*Gaeumannomyces graminis* var. *tritici*), eyespot caused by the sibling fungal species *Oculimacula acuformis* and *O. yallundae* and sharp eyespot caused by *Rhizoctonia cerealis*. *Fusarium* species and *S. nodorum* are also found on the stem-base. Several fungal pathogens attack the foliage, both biotrophs and necrotrophs. The rusts and powdery mildew (*Blumeria graminis*) are biotrophs, while the necrotrophs include leaf blotch diseases such as septoria tritici blotch caused by *Mycosphaerella graminicola* (anamorph *Septoria tritici*), tan spot caused by *P. tritici-repentis* (anamorph *Drechslera tritici-repentis*) and stagonospora nodorum blotch caused by *P. nodorum* (anamorph *S. nodorum*). Well-known ear diseases include glume blotch (*S. nodorum*), *Fusarium* spp., tan spot, some of the rusts and powdery mildew. Fungal pathogens can be found on the seed, in the soil or in the air, *i.e.* they are seedborne, soilborne or airborne. Typical seedborne pathogens have already been mentioned and among the soilborne are the many pathogens living on crop residues or having resting spores as part of their life cycle, such as *Claviceps purpurea*, *Fusarium*, *Gaeumannomyces graminis*, *Microdochium nivale*, *Oculimacula acuformis* and *O. yallundae*, *M. graminicola*, *P. tritici-repentis*, *P. nodorum, Rhizoctonia cerealis* and *Typhula* spp. Winter damage and outwintering are caused by low-temperature fungi such as *Microdochium nivale* causing snow mould and *Typhula* spp. causing speckled snow mould or typhula snow mould. Rain is an important factor in dispersal through rain-splash of some diseases such as eyespot, *Fusarium* spp., septoria tritici blotch, stagonospora nodorum blotch, while wind-dispersed rusts are also spread by rain splash (Fitt *et al.*, 1989; Geagea *et al.*, 1999).

It is not always easy to identify the cause of bad plant health. Symptoms of different biotic and abiotic stresses may look alike and usually more than one stress affects a plant. A good example is blotches or necrotic leaf tissue due to leaf blotch diseases (LBDs) on winter wheat caused by *M. graminicola* (anamorph *S. tritici*), *P. tritici-repentis* (anamorph *D. tritici-repentis*) and *P. nodorum* (anamorph *S. nodorum*). It is fairly easy to differentiate between the symptoms if only one of these fungi attacks the plant, but when more than one of fungal pathogens is present together with other stress factors it is almost impossible to quantify the attack of each single disease. To avoid this problem it is sometimes possible to use green leaf area as a measure (Nilsson, 1995; Bryson *et al.*, 1997; Ewaldz, 2000; Gooding *et al.*, 2000; Audsley *et al.*, 2005; Bancal *et al.*, 2007; McCartney *et al.*, 2007) and simply consider the three fungal species in combination, assessed as one disease and designated leaf blotch diseases (LBDs). However, new analytical methods, *e.g.* real-time PCR (polymerase chain reaction, in which a detectable segment of DNA is amplified) make it possible to identify and quantify single fungal species on a host, *e.g.* as recently used to clarify and distinguish between the sibling eyespot fungi and sharp eyespot at early growth stages (Henson & French, 1993; Lagerberg *et al.*, 1996; Martin *et al.*, 2000; Turner *et al.*, 2001; Guo *et al.*, 2007; Almquist *et al.*, 2008; Miller *et al.*, 2009; Munkvold, 2009).

2.3.2 Crop loss assessment

The ultimate importance of crop growth and yield has directed many researchers to the identification of yield constraints and crop loss assessment studies. Measurement of disease is fundamental in crop loss assessment studies (Chester, 1950; Large, 1966; Walker, 1983). Determination of the economic damage level (the level of attack at which the benefit of control just exceeds its costs) and the damage threshold, appropriate dose and precise timing of pesticides and the use of pesticides only when needed requires information from crop loss assessment studies. The measurement of crop losses reveals the decisive and disastrous effects of pests and diseases, at worst leading to famine and social catastrophes (Chiarappa *et al.*, 1972; James, 1974; Teng & Krupa, 1980; Chiarappa, 1981). Horsfall & Cowling (1978) listed a number of reasons why it is important to measure disease and the resulting crop losses, including:

- to form the basis for setting priorities in research, legislation and sales,
- to generate cost/benefit ratios, essential for decisions at several levels,
- to allow forecasts of crop production,
- to monitor the variable efficacy of resistant varieties and pesticides,
- to generate information to advisory and regulatory activities.

During the late 1970s and thereafter, improvements were made. Crop loss assessment developed its own terminology and mathematical and statistical models including Bayesian probability theory were introduced (Teng *et al.*, 1977; Teng & Krupa, 1980; Chiarappa, 1981; Zadoks, 1985; Nutter *et al.*, 1993; Cooke, 1998; Yuen & Hughes, 2002; Savary *et al.*, 2006; Milne *et al.*, 2007; Zhang *et al.*, 2007).

Crop loss estimations have been made for many diseases in winter wheat, supplemented with studies on how different factors affect the diseases and thereby the crop losses. These estimations have been made from the results of disease surveys, fungicide trials, variety trials and disease assessment on single tillers (Buchenau, 1975; Richardson *et al.*, 1976; King, 1977; Sim IV *et al.*, 1988; Rao *et al.*, 1989; Shaw & Royle, 1989; Cook *et al.*, 1991; Daamen & Stol, 1992; Jones, 1994; Jørgensen *et al.*, 1996; Hardwick *et al.*, 2001; Bancal *et al.*, 2007). The annual percentage yield losses have often been estimated using formulae from previous studies (Mundy, 1973; Scott & Hollins, 1974; King, 1976; Clarkson, 1981; Clarkson & Cook, 1983; Thomas *et al.*, 1989). Disease surveys are carried out annually in *e.g.* Denmark, England and Wales, the USA, the Netherlands and Sweden. The surveys differ to some extent, *e.g.* whether the sampling is carried out in a treated or untreated crop, but their objectives are similar and clear – to estimate the importance of different diseases and how and when to control them. Accordingly, large numbers of studies have been carried out, but only a few results from the consistent surveys in England and Wales are presented here. King (1977) recorded yield losses due to mildew, LBDs and yellow rust during 1970-1975, including eyespot in the last year. On average during the six years, mildew reduced annual yield by 2.9%, followed by LBDs (2.2%), yellow rust (0.2%) and, during the last year, eyespot caused 0.9% losses which was the second most damaging disease after mildew in that year. *S. nodorum* was the most widespread of the LBD pathogens during these years. However in a single year (1972) during this period with the most severe yield losses (7.4%) due to LBDs *S. tritici* was the most common foliar pathogen. At this time, yield loss due to LBDs was calculated on the basis of results from four field trials in which actual yield loss was approximately correlated to the severity on the flag leaf (leaf 1), but in a later equation Thomas *et al.* (1989) used the severity on leaf 2 x 0.42. For yellow rust the findings of Mundy (1973) and King (1976) were used when estimating the percentage yield loss (yield loss is percentage disease on flag leaf x 0.4). For eyespot the equation by Scott & Hollins (1974) was used; yield loss is 0.5 x the percentage incidence of severity on affected stems. During 1985-1989, a period of quite constant fungicide use and cereal husbandry in England and Wales, septoria tritici blotch was the most damaging disease, and explained 41% of total yield losses due to the three most damaging diseases, followed by eyespot (31%) and powdery mildew (28%) (Cook *et al.*, 1991). Considering septoria tritici blotch, eyespot and mildew together with *S. nodorum*, sharp eyespot, yellow rust and brown rust, these diseases explained 31, 23, 21, 14, 9, 1 and 1%, respectively, of the total yield losses during these five years. In addition, take-all caused a 6% yield reduction in second and third wheats. Hardwick *et al.* (2001) reviewed the results of the England and Wales disease survey, including analyses of major changes occurring during the ten-year period 1989-1998. Compared with the period 1985-1989 eyespot, septoria leaf blotch and powdery mildew still were the most damaging diseases (Cook *et al.*, 1991). LBDs, eyespot, mildew, glume blotch, sharp eyespot, yellow rust and brown rust were responsible for 30, 34, 25, 2, 6, 2 and 1%, respectively of the total yield losses during these ten years. These yield losses occurred in spite of considerable fungicide use, since more than 93% of the crops were treated and more than two applications per crop were given. Major changes during these ten years were the decline in powdery mildew from 1991 to 1998 due to more resistant varieties, glume blotch remaining at very low levels and the major foliar disease septoria leaf blotch showing large variations in disease severity between years (0.6 to 7.8% of samples affected on the second leaf). Eyespot, which was more severe than sharp eyespot, varied between 4.8 and 18.9% of stems affected by moderate to severe lesions (Scott & Hollins, 1974). These results from England and Wales are in agreement with earlier Swedish results from field trials carried out 1976-1992 in which leaf blotch diseases, eyespot plus other stem base diseases and mildew were the most important (Andersson *et al.*, 1986; Wiik *et al.*, 1995).

2.3.3 Disease cycles and epidemiology

A chain of events or interconnected stages of development has to occur before a healthy plant or crop becomes visibly diseased. These events or stages include the arrival of a pathogen to the host (inoculation), attachment to the host, recognition between host and pathogen, spore germination, appressorium formation, penetration, infection, colonisation, and dissemination, often by air and or water. Some diseases are monocyclic, with only one disease cycle per year, while others are polycyclic, with many disease cycles per year, the latter type causing rapid and explosive epiphytotics.

In epidemiology, different factors that affect the disease cycle and especially the rates of the events are studied (van der Planck, 1963;

Vanderplank, 1982, 1984; Zadoks & Schein, 1979; de Vallavieille-Pope *et al.*, 2000):

- the inoculum available at the beginning of the season,
- the length of the latent period (time from arrival of spores until formation of new spores),
- the relative rate of spore production,
- the length of the infectious period (the period of production of new spores or the sporulation)
- the effectiveness of inoculum.

Pathogens differ in respect of these five factors and how they are affected by their host and the environment (the disease triangle), *i.e.* population or disease dynamics and patterns in time and space leading to different life strategies (Kinkel, 1997; García-Guzmán & Morales, 2007). Some diseases such as the rusts can be called r-strategists, with a high inoculum level, short latent period, short infectious period, high rate of spore production and low effectiveness of inoculum. Other pathogens have the opposite strategy and are called k-strategists (Zadoks & Schein, 1979). Knowledge about the stages in the disease cycle is used in many plant disease prediction models, *e.g.* for *Fusarium* sp., *P. triticina*, *P. striiformis*, *B. graminis*, *S. tritici*, *S. nodorum* and *P. tritici-repentis* (De Wolf & Isard, 2007). The battle is won against monocyclic diseases such as common bunt (*T. tritici*) or dwarf bunt (*T. controversa*) if the initial inoculum on the seed and in the soil is removed or eradicated. For polycyclic diseases, such as many of those in wheat, the reduction of initial inoculum must be followed by disease rate-reducing control measures, *e.g.* race-nonspecific resistance or fungicides.

2.3.4 Variability and adaptation

The variability and adaptation of living organisms is striking. Specific pathosystems are the continuous outcome of co-evolution and man-guided evolution in agricultural systems and the step-wise evolution of virulence and resistance corresponding to compatible and incompatible reactions between plant host and pathogen. The gene-for-gene concept has been useful in plant breeding (Flor, 1971). McDonald & Linde (2002) conclude that pathogen populations with both sexual and asexual reproduction, large and viable populations (r-strategists), high mutation rate and extensive gene flow have evolutionary potential with good ability to overcome host resistance. Plant breeders compete with the pathogen to achieve a true and durable resistance by searching for new resistance, by using both R-genes and polygenic resistance, and by using different techniques such as tissue culture and genetic engineering. One example to improve host resistance involves pyramiding resistance genes (Pedersen & Leath, 1988). However, mutations, recombination and gene flow help the pathogens to adapt and to overcome the resistance, and enter or reenter the scene as a serious pathogen. In a similar way pathogen populations develop pesticide resistance (Horsten & Fehrmann, 1980; Dekker & Georgopoulos, 1982; Delp, 1986; Johansson & Wiik, 1989; Olvång, 1987, 1988; Metcalfe *et al.*, 2000; Bryson *et al.*, 2006; FRAC, 2009).

2.4 Control

Different means of production are used to produce grain yields of winter wheat of high quantity and the required quality. As already shown, the increase in yield per hectare has been striking since the 1960s, attributed to the introduction of fertilizers and pesticides, plant breeding and progress in crop husbandry. The use of pesticides is huge today and is increasing by 14% per year world-wide due to an increase in developing countries, but with a decline in the United States and Europe (Agrios, 2005). During 1999, 2.6 billion kg active ingredients were used world-wide, at a cost of about € 10 kg^{-1}. When pesticides are being approved for use, tests on their efficacy against pest and diseases and possible phytotoxic effects are essential, but investigations on their fate in the environment and health effects such as persistence and toxicity are also demanded by the authorities. Due to their properties pesticides have adverse effects on the environment and on different life forms, and should be used with care or not at all if other rational control measures are available. The use of pesticides is not the only approach to decrease the impact of plant diseases in agroecosystems. Several other control methods are available, both those that are preventative and those that are used directly or in acute situations. The control inputs may differ on how they influence epidemiological parameters, but they all limit disease development. Regulatory and preventative control methods may prevent the pathogen reaching its host by quarantine and other means of avoidance, by using pathogen-free propagation materials, *e.g.* seed, and by using resistant varieties. Direct and acute methods include biological, physical and chemical means of control, *e.g.* fungicide use.

2.4.1 Fungicides

Fungicides have become an essential and main control measure against plant diseases during recent decades (Knight *et al.*, 1997). A fungicide kills or inhibits the growth of a fungus or a number of fungi. A fungicide can also promote a fungus other than the target fungus or fungi, and can have other iatrogenic or unfortunate side-effects (Griffiths, 1993). For example, grain samples taken from untreated and fungicide-treated plots in Swedish field trials and stored for 6 months were analysed by the blotter paper method and by the osmotic method for *Septoria* (*Stagonospora*) spp., *Fusarium* spp. and *Drechslera* spp. Fungicide treatments had good efficacy against *Septoria* spp., the target fungus of the treatments, but in a few field trials the seedborne inoculum of *Drechslera* spp. and *Fusarium* spp. on the grain was significantly higher in field plots treated with fungicides than in untreated plots. Due to competition between pathogens, the place not occupied by *Septoria* spp. was probably utilised by *Drechslera* spp. and *Fusarium* spp. (Wiik, 1985; Fitt *et al.* 2006).

Several different types of fungicides are in use, *e.g.* the strobilurins, triazoles and benzimidazoles (Jenkins & Lescar, 1980; Davidse & de Ward, 1984; Schöfl & Zinkernagel, 1997; Bartlett *et al.*, 2002; Guo *et al.*, 2007). Fungicides can be classified according to:

- whether they are broad-spectrum, *i.e.* affect many fungal pathogens, or whether they are more selective,
- whether they are contact, translaminar or systemic (how they perform on or in the plant),
- whether they need to be applied in advance of the pathogen (protectants) or whether they destroy the pathogen when infection has already taken place (eradicants),
- their mode of action, not least of interest when discussing fungicide resistance.

The activity of fungicides depends not only on the active ingredient but also on the other ingredients included in the formulated product, such as wetting agents, emulsifiers and stickers. Much can be said about fungicide use, but here brief answers are given to the questions posed in the introduction: whether, why, where, which, when and how.

Whether? The question of whether or not to use a fungicide is not easy to answer, as fungicide use is an investment for the often unpredictable future, *i.e.* the grain yield gain at harvest and the profit of that extra grain yield. It has been shown in the evaluation of many field trials that fungicide input is too often not profitable for the farmer. In deciding whether to use a fungicide, the expected yield loss, both in terms of quantity and quality, must be known and the predicted yield gain due to treatment in a specific field, because a number of factors decide the outcome of a treatment. The cost-effectiveness of the treatment, the risk-awareness of the farmer, *etc.*, must also be known, so it is obvious that the answer to ‘whether?’ requires a lot of background data and knowledge.

Why? Why fungicides are used has already been revealed – it is because farmers, plant protection companies and consumers make profits or savings due to the demand and the marketable value of the extra quantity and quality of grain yield attained by fungicide use. Due to the importance of fungicides in the USA Gianessi & Reigner (2006) proposed policies to protect fungicides.

Where? Fungicides are used in crops/varieties and regions with recurrent and frequent problems with specific diseases and sometimes as a safeguard against diseases that may perhaps come, *e.g.* the recurrent attacks of LBDs and occasional yellow rust attacks in winter wheat in NW Europe.

Which? A lot of field trials are carried out in many countries to show the performance and benefit of different fungicides. It is important to choose a fungicide that is effective against the prevailing fungal pathogens, *e.g.* choosing strobilurins instead of conventional fungicides at that time gave higher yields in winter wheat varieties (Bayles, 1999). During the first part of the 1980s a new broad-spectrum fungicide (Tilt 250 EC, a.i. propiconazole) for winter wheat was introduced onto the market in Sweden as the only fungicide now needed, but this fungicide proved not to be effective against all diseases present. In the year of introduction (1983), eyespot and stem-base diseases were a serious problem and a number of farmers were disappointed by crop lodging and insufficient effects of the novel fungicide. This incident stresses the importance of the appropriate choice of fungicide or fungicide mixture, which is much more comprehensible today than some decades ago (HGCA, 2009; SJV, 2009), but it is not possible to be prepared for every disease that might become a problem in a crop. It is also important to choose the appropriate dose. A number of field trials have studied the dose-response between the fungicide and relevant diseases (Ewaldz, 2000; Paveley *et al.*, 2000; Paveley *et al.*, 2001; Paveley *et al.*, 2003; Mercer & Ruddock, 2005; Lockley & Clark, 2005; Knight *et al.*, 2008; Lockley *et al.*, 2008; Bürger *et al.*, 2008). An adaptation of the dose to the leaf area is also an option, as presented for apple trees and in viticulture (Walklate *et al.*, 2003; Siegfried *et al.*, 2007).

When? The timing of application of a fungicide depends on many factors such as when the pathogen occurs and the parts of the crop that need to be protected. Results from field trials and modelling have contributed to knowledge of the best application time(s) (Cook, 1977, 1980, 1987; Lipps & Madden, 1989; Cook *et al.*, 1999; Nicolas, 2004; Parsons & Te Beest, 2004). Disease scouting and thresholds such as the economic damage level or the intensity of the disease attack may be useful when a decision is to be taken on whether a fungicide should be used (Onstad & Rabbinge, 1985;

Zadoks, 1985; Pedigo *et al.*, 1986; Emmerman *et al.*, 1988; Wiik, 1993; Hansen *et al.*, 1994; Larsson, 2005). Even better are decision support systems (DSS) integrating factors decisive for impending disease development such as properties of the variety, soil and plant status, sowing time, crop rotation, fertilizers applied, the actual attack, weather prognosis, *etc.* (Stynes & Veitch, 1983; Yarham, 1988; Cook & Thomas, 1990; Stevens *et al.*, 1997; Colbach & Saur, 1998; Hall & Sutton, 1998; Paveley, 1999; Smith & Gooding, 1999; Hardwick *et al.*, 2001; Audsley *et al.*, 2005; Milne *et al.*, 2007; Zhang *et al.*, 2007; Burke & Dunne, 2008; Loyce *et al.*, 2008).

How? How a fungicide is used is important because misuse can promote fungicide resistance, *e.g.* if fungicides with the same mode of action are used repeatedly. The risk of impaired efficacy of a fungicide arises due to the properties of both the fungicide and the pathogen (Dekker & Georgopoulos, 1982; Staub, 1991; Bryson *et al.*, 2006; FRAC, 2009). For two very important groups of fungicides – the strobilurins and the azoles –widespread resistance and reduced sensitivity of *S. tritici* is now a fact (Fraaije *et al.*, 2003, 2005; Brunner *et al.*, 2008). The demand for varieties resistant to septoria tritici blotch and fungicides with a new mode of action will almost certainly increase (Arraiano *et al.*, 2009).

2.4.2 Host plant resistance

Fortunately, all plants are not affected by all diseases since they have nonhost resistanceto most pathogens, but enough fungal pathogens exist to pose a threat to many agricultural crops. However, resistance in host plants can be exploited and used, *e.g.* by breeding winter wheat varieties with resistance against yellow rust and powdery mildew. The durability of host resistance differs due to resistance type. If there is only one or a few resistance genes in the plant host, the plant has monogenic, R-gene, vertical or race-specific resistance. It is usually easy for some pathogen populations to adapt and overcome monogenic race-specific host plant resistance. A way to strengthen the race-specific resistance is to include or pyramid more than one race-specific resistance gene in a variety. This is a complicated process (Pedersen & Leath, 1988) although now possible with the help of QTL mapping (Bagge *et al.*, 2008). A plant may have more general resistance depending on many genes, *i.e.* polygenic, quantitative, adult-plant, horizontal or race-nonspecific resistance. A combination of race-nonspecific and race-specific resistances will probably give the most effective and durable resistance.

There is no doubt that the value of varieties bred for disease resistance is very high (McDonald *et al.*, 1971; Priestley & Bayles, 1988; Hogenboom, 1993; Smale *et al.*, 1998; Bockus *et al.*, 2001; Marasas *et al.*, 2003). As already mentioned, catastrophic epiphytotics due to lack of resistance in combination with suitable weather for a pathogen have resulted famine, human disease, death and emigration. Even when not causing catastrophes, lack of resistance against prevailing diseases constrains annual crop yields worldwide. Good recent examples are host resistance to *S. tritici*, which decreased in varieties grown during the early 1980s whereupon septoria tritici leaf blotch dramatically increased in the UK, wheat yellow rust adaptation to race-specific resistance leading to the 'breakdown' of resistance and yellow rust epiphytotics, and more severe attacks of powdery mildew attributed to the use of a higher proportion of susceptible varieties (Bayles, 1991, 1997; Bayles *et al.*, 2000; Mesterházy *et al.*, 2000; Hardwick *et al.*, 2001).

International and national programmes contribute to plant breeding successes. Anticipatory breeding covers annual pathogenicity surveys or pathotype surveillance, identification and characterisation of host resistance and enhancement service to breeders and cultivar replacement and recommendations, *e.g.* resistant cultivars in rust-prone areas (McIntosh & Brown, 1997; Hovmøller & Henriksen, 2008). However, a disease not present or not discovered in surveys may later become an important disease. A disease can be temporarily absent for many years but revive when new popular varieties without the essential host plant resistance are introduced or when other disease-suppressing factors are discontinued (Johnson, 1992). The International Maize and Wheat Improvement Center (CIMMYT) has had an immense influence on world-wide wheat breeding, in which host plant resistance only is one part. Breeding material is tested at locations all over the globe in environments differing in moisture, temperature and so forth. Black stem rust, yellow rust and brown rust, foliar diseases such as *Septoria* spp. and tan spot, root diseases and Fusarium head blights have high priority (Ortiz *et al.*, 2007).

North America has a long tradition in host plant resistance breeding against rust diseases, which was started to cope with the devastating epiphytotics that occurred from time to time. Whether a year became remembered as one with a devastating epiphytotic depended on the outcome of interactions between crop maturity, availability of primary inoculum in the spring, long distance dispersal, time of infection, rate of disease development, environment and host resistance – and we still live with this uncertainty (Roelfs, 1988, 1989; Eversmeyer & Kramer, 2000; Line, 2002; Chen, 2005, 2007; Kolmer *et al.*, 2007; Milus *et al.*, 2009). The 'boom and bust' cycle of cereal rust resistance genes and the contest between fungal pathogens and plant breeders is experienced and anticipated in the huge acreage of wheat in the Great Plains of the USA and the breeders have been successful frequently, but far from always (Chen, 2005). During recent years, epiphytotics of yellow rust in 'new' territories have again challenged researchers to breed for resistant varieties with durable resistance – 62 new races of *P. striiformis* f. sp. *tritici* have been identified since 2000 (Chen, 2007). In addition, these 'new' isolates are more aggressive and adapted to higher temperatures (Milus *et al.*, 2009). Brown rust is the most common of the rusts on wheat in the USA, probably due to the high variability in *P. triticina* populations. New varieties probably have to combine race-specific and race-nonspecific resistance against brown rust to be durable (Kolmer *et al.*, 2007). Even if black stem rust has not been a problem in the USA since the epiphytotics of the 1950s continuous surveys are made of both domestic and foreign *P. graminis* populations to reveal changes that may threaten wheat production (McVey *et al.*, 2002; Jin *et al.*, 2007).

In Australia, plant breeding to control stem rust and leaf rust started 90 years ago, but with increased activity after a severe stem rust epiphytotic in 1973 (McIntosh, 2007; Park, 2008). Yellow rust appeared for the first time in 1979 (Wellings, 2007). Some highlights of the Australian Cereal Rust Control Programme today are how to achieve durable resistance, *e.g.* by marker-assisted selection, and triple rust resistance in wheat (Bariana *et al.*, 2007; Ellis *et al.*, 2007).

In Europe, breeding for host plant disease resistance has a long history, including in Sweden (Nilsson-Ehle, 1904; Åkerberg, 1986; Lundin, 1973, 1997). Botanist and geneticist Sir Rowland Harry Biffen at Cambridge realised that Mendel's law of inheritance could improve plant breeding. Herman Nilsson-Ehle, the first professor in genetics in Sweden and a contemporary of Biffen, also applied Mendel's law of inheritance in his work at Lund University and the Swedish Seed Association. Nilsson-Ehle drew attention to the great importance of plant diseases and host plant disease resistance in a lecture at Svalöv in 1904 (Nilsson-Ehle, 1904; Åkerberg, 1986):

"*Perhaps the majority consider the problem of plant disease has but little to do with breeding work here at Svalöf but upon closer consideration it will be found that the opposite is the case and that they have the most intimate relations with each other. It is hardly possible to control many diseases in any other way than by means of breeding resistant varieties.*"

The whole speech, published in the Journal of the Swedish Seed Association, is foresighted and in spite of the hundred years since then it is still of interest.

Yellow rust – "the severest of the rust species in Sweden which has long been known as one of our most difficult plant disease" according to Nilsson-Ehle (1904) – is an example of a disease that negatively influenced yield at the turn of nineteenth century, a period when yellow rust epiphytotics severely attacked the crop after mild winters every three or four years (Jönsson, 1978; Lundin, 1997). The Nilsson-Ehle variety Pansar was resistant for a few years before new races of yellow rust brought about the 'breakdown' of the resistance, but the Swedish varieties Standard and Saxo bred by Birger Kajanus and Sven Otto Berg had more durable resistance and later crossings with these varieties made yellow rust a less serious problem as long as Swedish varieties (except Sleipner and to some extent Holme) were dominant in Sweden (Jönsson, 1978; Lundin, 1997). In 1972 the German variety Kranich, high-yielding at that time, was grown in southern Sweden and on a significant acreage in Denmark. A new race of yellow rust attacked Kranich and a mild winter favoured disease development (Andersson, 1973). Although the relative lateness of the attacks in Sweden restricted yield loss, in Denmark the situation was aggravated by earlier attacks on a large acreage of Kranich and the variety Cato, which also became susceptible. Again, the vulnerability of using narrow host plant resistance against serious potential diseases was confirmed.

The Swedish variety Sleipner was not grown much in Sweden but in the UK the variety once occupied more than 20% of the national acreage. The 1989 yellow rust epiphytotic was largely associated with this variety, see Figure 6 showing the total adaptation during 1989 of the yellow rust population to Slejpner (WW 78263) with race-specific gene Yr9 (Bayles *et al.*, 1990).

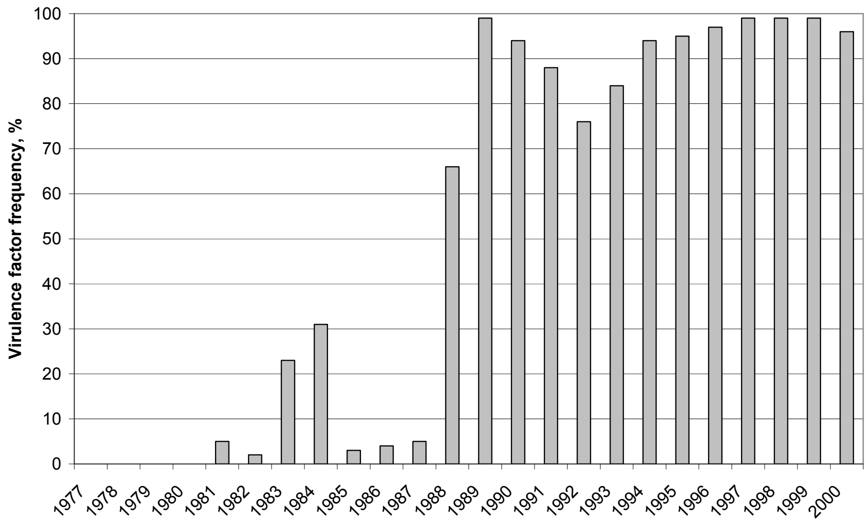

Figure 6. Virulence factor frequency (%) (WYV9) in the UK against wheat yellow rust resistance gene Yr 9 during 1977-2000. Compiled from Bayles *et al.* (1990) and Bayles & Stigwood (2001).

The importance of disease surveys

Surveys of plant diseases, which are useful for farmers and breeders, are performed in several European countries, continuously or for a shorter time, often starting after severe epiphytotics. After a severe black stem rust epiphytotic in wheat in Sweden in 1951, a survey of pathogen variability of cereal rusts started to give breeders information to improve disease resistance breeding (Leijerstam, 1961). In addition, during this time surveys on powdery mildew started in cooperation with other European countries (Leijerstam, 1972). In Denmark similar work was done (Hermansen, 1968; Hermansen *et al.*, 1978). Powdery mildew attracted much attention for a long time as it overtook yellow rust as the dominant cereal disease in Sweden (Lundin, 1997). Leijerstam continued his research on powdery mildew (Leijerstam, 1972) and investigations were later carried out in Denmark on Scandinavian wheat varieties (Hovmøller, 1989). This kind of work must be respectfully acknowledged, *e.g.* Leijerstam (1972) tested 39 powdery mildew isolates on a wheat collection of 6700 hexaploids and 3400 tetraploids to detect sources of resistance. During the 1980s and 1990s virulence surveys were made with a wind impaction spore trap (WIST) (Wolfe *et al.*, 1981) complemented with the exposure of mobile nurseries at a few locations in southern Sweden (Wiik, 1991). Swedish surveys and similar research from several other European countries were continued, e.g. in COST Action 817 on 'Population studies of airborne pathogens on cereals as a means of improving strategies for disease control' 1993-1999, a cooperative effort of European countries (Wolfe & Limpert, 1987; Helms Jørgensen, 1991; Limpert *et al.*, 1996). In a special issue of Agronomie, results from COST 817 that are valuable for disease resistance breeding and deployment of host resistance against powdery mildew and rust were presented (Østergård, 2000).

A good example of a European survey is the UK Cereal Pathogen Virulence Survey (UKCPVS), started in 1967 after an unexpected yellow rust epiphytotic due to the adaptation of the rust population to a widely grown and previously resistant wheat cultivar (HGCA, 2009; NIAB, 2009). A lot of samples collected all over the country are each tested on a disease-specific set of differentials, *i.e.* a set of cultivars or lines with known race-specific genes, which makes it possible to classify the virulence of a sample (*e.g.* WYV9, Figure 6). Based on the results from the surveys, sometimes with diversification schemes, the advisory service can recommend and farmers can choose appropriate varieties that contribute to disease control and maintenance of host resistance and give valuable information to breeders (Wolfe & Schwarzbach, 1975; Priestley, 1981; HGCA, 2009; NIAB, 2009). During recent years valuable contributions have been made to understanding host-pathogen interactions in the global pathosystem of wheat yellow rust. The results show rapid adaptation, increased aggressiveness, fast and intercontinental long dispersal spread (Boshoff *et al.*, 2002; Brown & Hovmøller, 2002; Wellings, 2007; Chen, 2005; Hovmøller *et al.*, 2008) and thus challenge the plant breeder to find useful resistance against yellow rust even faster than before (Johnson, 1988; Hovmøller, 2007).

Strategies for deploying resistance

Systems of deploying and managing disease resistance genes to reduce losses from epiphytotics have been proposed, in particular to stop devastating epiphytotics like the stem rust epiphytotic in the USA in 1953/1954, the brown rust epiphytotic in Mexico in 1976/1977 and the brown rust epiphytotic in Pakistan in 1977/1978. One way to cope with plant disease is to use multilines, a variety composed of many more or less defined lines, *e.g.* differing in race-specific resistance against one of the rusts – a system with interchangeable isolines (Browning & Frey, 1969; Borlaug, 1981; Priestley, 1981). In some countries the use of different R-genes on farms or within a region is recommended, agreeing with the above-mentioned diversification schemes (Priestley, 1981; Finckh & Wolfe, 1998; HGCA, 2009). The use of variety mixtures has been proposed, especially for airborne pathogens, but not much used with the exception of some countries, e.g. in Eastern Europe during the Cold War period (Finckh *et al.*, 2000; Wolfe, 2000). Mundt *et al.* (1995a and 1995b) demonstrated reduced attacks of LBDs (*S. tritici* and *S. nodorum*) and yellow rust in cultivar mixtures compared with their component pure stands, but the reductions were usually quite small. However, the mixtures improved yield stability relative to the pure stands.

The increase in *S. tritici* during the 1980s in the UK and other countries was partly due to growing more susceptible varieties than previously used (Bayles, 1991). Until the beginning of the 1970s there were few published investigations on sources of resistance to *S. tritici* (Rosielle, 1972). Factors that make progress slow in resistance breeding against *S. tritici* were identified, such as the great variability in *S. tritici*, long latent period of the disease, variability in symptom expression, unspecified environmental conditions during tests, unclear breeding strategies and lack of uniform methodologies (Eyal, 1999a; Goodwin, 2007). Thirteen R-genes are known for resistance to septoria tritici blotch, but their contribution as well as that of polygenic resistance is not fully known (Eyal, 1999a; Arraiano *et al.*, 2009). In more than 7 500 accessions, Rosielle (1972) found more resistant varieties in *T. durum* than in *T. aestivum*; 34 selections of *T. aestivum* and 266 of *T. durum* showing reliable expression of resistance. Varieties in 20-30 naturally infected variety trials in the UK showed significant differences against septoria tritici blotch, with mean infection levels ranging from 3.8-9.4% (Bayles *et al.*, 1985). Some dwarfing genes appeared to influence the level of resistance to *S. tritici* (Baltazar *et al.*, 1990). Research is underway to find new sources of resistance and the results so far look promising. Pyramiding resistance genes is also an option against septoria tritici blotch (Zhang *et al.*, 2001; Eriksen *et al.*, 2003; Chartrain *et al.*, 2004; Arraiano & Brown, 2006). Furthermore, traits that confer tolerance – the disease is tolerated by the plant without giving reduced yield – and escape provided by plant morphological traits might be important in the search for durable resistance (Arraiano *et al.*, 2009; Bingham *et al.*, 2009). Whether *S. tritici* follows a gene-for-gene relationship has been debated from time to time and such knowledge is useful when identifying pathogenicity and resistance variation (Eyal *et al.*, 1985; Van Ginkel & Scharen, 1988; Johnson, 1992; Kema *et al.*, 1996; Kema & van Silfhout, 1997). Use of resistant varieties provides benefits if fungicides and resistant varieties are interchangeable with each other, which has been shown in the potato-late blight pathosystem and later in the wheat-septoria tritici blotch pathosystem (Fry, 1978; Stevens *et al,*. 1997).

Resistance against eyespot was low in most varieties in the beginning of the 1980s. An exception was the French variety Cappelle-Desprez, but it was not sufficiently resistant under severe attacks (Doussinault *et al.*, 1983). Benzimidazole fungicides were used against eyespot but fungicide resistance and control failures increased the efforts to search for host plant resistance, although difficulties due to undesirable traits made breeding against eyspot difficult (King & Griffin, 1985). There are now new fungicides that are effective against eyespot, such as prochloraz of the chemical class azole and cyprodinil of the chemical class pyrimidine. The latter is more effective but fungicide resistance may also evolve for both of these types of fungicides, and resistant varieties are preferred to fungicides in many ways (Leroux & Gredt, 1997; Babij *et al.*, 2000; Bateman *et al.*, 2000; Ray *et al.*, 2004). Cappelle-Desprez was used extensively in the UK for more than 20 years, probably due to its resistance against eyespot (Hollins *et al.*, 1988). The variety Rendezvous with eyespot resistance from *Aegilops ventricosa* performed better than varieties with resistance from Cappelle-Desprez in field trials over six years in the UK but most other varieties were susceptible (Doussinault *et al.*, 1983; Jones, 1994). For now, only four genes for eyespot resistance have been described; gene *Pch1* derived from a wild wheat, the most effective and widely used but variable, gene *Pch2* in the variety Cappelle-Desprez, and resistances from two diploid wheat species, *Pch3* derived from the mosquitograss (*Dasypyrum villosum*) present in Pennsylvania USA, and a gene from goatgrass (*Aegilops tauschii*) (Murray *et al.*, 1994; Jones *et al.*, 1995; Yildirim *et al.*, 1995; Lind, 1999; Leonard *et al.*, 2008). The eyespot-resistant varieties Madsen and Hyak with the *Pch1* gene were introduced in the Pacific Northwest of the USA during the late 1980s. These varieties became very popular because of their eyespot resistance and many growers were able to avoid fungicide treatment by choosing them. In recent years new resistance to eyespot has been found in einkorn (*T. monococcum*), resistance that is more effective than the genes *Pch1* and *Pch2* in hexaploid wheat, in tall wheatgrass (*Thinopyrum ponticum*) and intermediate wheatgrass (*Thinopyrum intermedium*) (Cadle & Murray, 1997; Li *et al.* 2004, 2005). Pyramiding resistances from gene *Pch1* with *Pch2* will probably increase the resistance against eyespot, *i.e.* resistance at both the seedling stage and the adult stage (Muranty *et al.*, 2002). Development in both the isoenzyme marker technique and DNA-marker assisted selection of eyespot resistance will hopefully contribute to producing varieties with durable resistance against eyespot in the near future (Chapman *et al.*, 2008).

Swedish variety trials

In the Swedish variety trials, the severity of LBDs, brown rust, yellow rust and powdery mildew is assessed on the upper leaves (Table 1). Brown rust, yellow rust and powdery mildew differ between varieties, as do the LBDs. Larsson *et al.* (2005) point out that earlier varieties probably get higher severity ratings due to the fact that the assessments are made on the same day in all varieties, disregarding differences in maturity, and to the fact that a single variety is not always represented in all field trials (Table 1). In addition, plant height contributes to escape (Arraiano *et al.*, 2009). Nevertheless, variety trials give indications of differences between varieties. Among five commonly grown varieties Ritmo is attacked more than any other by powdery mildew and LBDs and Kosack by brown rust. Ritmo is attacked by yellow rust, but yellow rust is a rare pathogen and few assessments have been made (Larsson *et al.*, 2008). The fodder wheat variety Marshal gave a higher yield than Kosack, both in untreated and fungicide-treated plots. Marshal was attacked by mildew to the same level as Ritmo, but Ritmo did not respond to fungicide treatment as positively as Marshal. These figures do not satisfactorily explain the yield differences between the varieties. Variation in yield response by varieties in field trials can be in accordance with disease ratings, but the effect of a disease on yield in the field is probably best shown with isogenic lines and by untreated and selective fungicide-treated plots (Seck *et al.*, 1988; Cook & Thomas, 1990; Line, 2002).

Table 1. *Disease incidence in untreated plots, plant height, maturity and relative yield in untreated and fungicide-treated plots for five winter wheat varieties in field trials carried out during 2000-2004 in Sweden (Larsson et al., 2005)*

Variety	LBDs	Brown rust %	Yellow rust %	Powdery mildew	Plant height	Maturity days	Relative yield to Kosack[c] in plots	
	%	%	%	%	Cm	days	untreat.	treat.
Kosack	26	8	0	4	105	327	100	100
Ritmo	37	6	3	10	78	323	99	105
Kris	30	2	0	5	75	324	106	109
Marshal	29	1	0	9	75	323	108	112
Bill	29	1	1	5	78	323	100	100
Highest value[a]	37	8	3	10	105	-	108	112
Lowest value[b]	22	1	0	2	73	-	97	99

[a] Most severe attack in any of 13 varieties.

[b] Least severe attack in any of 13 varieties.

[c] Relative yield in untreated and fungicide-treated plots.

2.4.3 Cultural methods

Introduction

Resistant cultivars and fungicides are effective control methods against fungal pathogens. However, cultural methods must not be forgotten. Sometimes the use of host resistance is included in cultural methods, but not here. Cultural methods are important for disease control in different ways. Pathogens can be avoided, *e.g.* by using seeds free from seedborne diseases. Biotrophs can be bypassed by avoiding crops that give rise to the green-bridge phenomenon. Pathogens with alternate hosts can be controlled by eradication or control of the alternate host. Soilborne inoculum on infected plant debris can be reduced, removed or biodegraded by soil cultivation and catch crops. Adjustment of host density by seed rate and fertilizer dose can restrict and promote disease development due to the establishment of different environments in thinner or denser crops. Crop rotation or crop sequencing reduces the build-up of fungal pathogens. Monocultures or cereal-dominated rotations can establish life-sustaining conditions for some plant pathogens. Actions or agricultural practices that are carried out earlier or later than usual can restrict growth of fungal fungal pathogens and thereby limit plant disease development similar to that of fungicides and host resistance. The use of cultural methods often is a compromise, *e.g.* between disease suppression and lower yield. Agricultural practices favouring epidemics and cultural practices to control plant diseases have been reviewed by Cowling (1978) and Yarham (1988).

Soil pH

Cephalosporium stripe (caused by *Cephalosporium gramineum*) increased with increased soil pH (Love & Bruehl, 1987). Take-all (caused by *Gaeumannomyces graminis*) were reduced by lowered soil pH due to ammonium fertilization (Smiley & Cook, 1973).

Nutrients – nitrogen, phosphorus, potassium and silicon

Fertilizing with nitrogen, especially early application, favour many diseases such as powdery mildew, septoria tritici blotch, stagonospora nodorum blotch, brown rust (Johnston *et al.*, 1979; Broscious *et al.*, 1985; Howard *et al.*, 1994; Leitch & Jenkins, 1995; Jørgensen *et al.*, 1997; Olesen *et al.*, 2003; Simón *et al.*, 2003; Neumann *et al.*, 2004). In a field trial located in a field with cereal-dominated crop rotation in south-western Sweden in 2002-2004 grain nitrogen use efficiency was larger with crop protection than without, resulting in larger yields (Delin *et al.*, 2008). Interactions between nitrogen

level and the attack of pests and diseases are well-known, *e.g.* demonstrated in field trials with increasing inputs of pesticides, nitrogen and growth regulators during 1978-1985 in southern Sweden (Andersson *et al.*, 1986) and in Croatia in 2000-2002 (Varga *et al.*, 2005). Application of ammonium decreased the levels of eyespot (Colbach & Saur, 1998).
Cunfer *et al.* (1980) demonstrated that glume blotch (caused by *S. nodorum*) increased with increased phosphorous application. Foliar sprays with potassium chloride (KCl) fertilizers reduced powdery mildew and septoria tritici blotch (Cook *et al.*, 1993). Both phosphorus and potassium suppressed brown rust, but the effect of potassium might have been related partially to the chloride in the potassium chloride fertilizer (Sweeney *et al.*, 2000). Mann *et al.* (2004) demonstrated effect of foliar-applied potassium chloride fertilizer on septoria tritici blotch on the upper leaves but not on the lower, suggesting a contact activity. The reduction of powdery mildew by foliar application of a potassium chloride solution was hypothesized to be due to an osmotic effect on spore germination (Kettlewell *et al.*, 2000). Silicon is supposed to activate defence reactions against powdery mildew (Bélanger *et al.*, 2003). Under high disease pressure silicon increased plant resistance to powdery mildew, stagonospora nodorum blotch and septoria tritici blotch (Rodgers-Gray & Shaw, 2004). However, so far fertilizers have not contributed very much to plant disease control and much of the results reported are contradictory (Walters & Bingham, 2007).

Crop rotation

Take-all can be managed by non-cereal crops (at least a two-year break) in the rotation (Polley & Thomas, 1991; Hardwick *et al.*, 2001; Cunfer *et al.*, 2006; Sieling *et al.*, 2007; Fernandez *et al.*, 2009). Severity of take-all and incidence of *Fusarium avenaceum* increased with higher frequencies of grass and cereals in the crop rotations (Hall & Sutton, 1998). Wheat as pre-crop significantly increased the severity of take-all (Sieling *et al.*, 2007). Short crop rotations with only one break crop controlled tan spot under certain conditions (Bockus & Claassen, 1992). In Canada, the septoria disease complex (stagonospora nodorum blotch and septoria tritici blotch) in spring wheat was controlled by crop rotation (Pedersen & Hughes, 1992). In conditions favourable for disease development two years between wheat crops were needed, but in conditions unfavourable for disease development one year was sufficient. Bailey *et al.* (2001) found crop rotation to have little impact on disease relative to environment but crop rotations with non-cereal crops reduced septoria tritici blotch and stagonospora nodorum blotch compared with wheat after wheat. *Septoria* spp. severity tended to be greater in wheat following cereals (King, 1977).

Sowing date, date of heading, plant density

Early sowing increased the incidence of eyespot (Polley & Thomas, 1991; Colbach & Saur, 1998; Hardwick *et al.*, 2001). Early sowing and high plant densities resulted in higher incidences of powdery mildew (Jørgensen *et al.*, 1997). Tavella (1978) demonstrated a negative relationship between septoria tritici blotch and days to heading, *i.e.* earlier heading resulted in more septoria tritici blotch than later heading. Septoria tritici blotch decreased with later sowing (Gladders *et al.*, 2001). Days from sowing to heading was positively correlated to septoria tritici blotch severity recorded during a 38-year period in Australia (Murray *et al.,* 1990). They suggested that early sowing favours early establishment of septoria tritici blotch leading to more disease later on. Yellow rust was more severe in early-sown crops (King, 1977). Yellow rust increased with later sowing, but when adjusted for the effect of cultivars it was no longer significant (Gladders *et al.*, 2007). Earlier sown crops gave higher yield increases due to early fungicide treatment than later sown crops (Cook & Thomas, 1990). High seeding rates and narrow row spacing created a microclimate favouring the development of stagonospora nodorum blotch and septoria tritici blotch (Tompkins *et al.*, 1993).

Pre- and post-harvest practices

Incorporating straw reduced septoria tritici blotch, powdery mildew, brown rust and foot rot caused by *Fusarium* spp. (Rodgers-Gray & Shaw, 2000). Reduced tillage increased *Fusarium* spp. on wheat roots (Bailey *et al.*, 2001). Ploughing reduced tan spot relative to other tillage practices (Bockus & Claassen, 1992). Reduced tillage has many advantages but at the same time some diseases are favoured. Many diseases can be controlled by crop rotation (Bockus & Shroyer, 1998).

Identification and integration of cultural methods

Cavelier *et al.* (1998) used multivariate methods to analyse data from almost 1000 fields to study the relationships between cultural practices and the incidence of foot and root disease complex on cereals. They found crop rotation and excess of phosphate fertilizer to be an important factor to reduce eyespot, but not delaying the sowing date. Jeger (2004) pointed out that models evaluating disease management practice still had made no major impact in agriculture. Ennaïfar *et al.* (2007) defined 16 models to help farmers to improve take-all control. They found strong effect of soil texture on take-all.

2.5 Fungal diseases in focus

2.5.1 LBDs with the focus on septoria trititci blotch

The pathogens

The disease complex LBDs include three leaf blotch diseases; septoria tritici blotch caused by *Mycosphaerella graminicola* (anamorph *Septoria tritici*), stagonospora nodorum blotch caused by *Phaeosphaeria nodorum* (anamorph *Stagonospora nodorum*) and tan spot caused by *Pyrenophora tritici-repentis* (anamorph *Drechslera tritici-repentis*). During recent decades septoria tritici blotch has been the major wheat disease in Europe and some other countries (Eyal, 1999b) and the focus in this short summary is on septoria tritici blotch, but both tan spot and stagonospora nodorum blotch are potential diseases that might become widespread and destructive in time to come. Tan spot is quite often a severe disease in two-year and three-year wheats. Stagonospora nodorum blotch can easily be found using PCR methodology, whereas visual assessments may only show septoria tritici blotch (Almquist *et al.*, 2008).

Figure 7. Septoria tritici blotch. Photo: Peder Waern.

M. graminicola, the sexual stage of *S. tritici*, was found quite late (Sanderson, 1972, 1976; Scott *et al.*, 1988) and consequently its importance in plant disease epidemiology was recognised late (Shaw and Royle, 1989;

Hunter *et al.*; 1999; Zhan *et al.*, 2007). As leaf blotch is such an important disease on wheat world-wide (King *et al.*, 1983), knowledge of its biology is of the utmost importance. Recent literature on the pathogen causing septoria tritici blotch is now extensive, *e.g.* on genetics, taxonomy, evolutionary potential and coexistence (Cunfer & Ueng, 1999; Eyal, 1999b; McDonald & Linde, 2002; Palmer & Skinner, 2002; Fitt *et al.*, 2006). Sexual reproduction operates during the growing season and probably makes the pathogen more capable of overcoming host resistance (Zhan *et al.*, 2007). The life cycle of the anamorph *S. tritici* and teleomorph *M. graminicola* and host-pathogen relationships have been studied in many countries, but still several aspects are still not fully understood (Eyal, 1999b; Hunter *et al.*, 1999; Palmer and Skinner, 2002). In Sweden the knowledge of epidemiology, dormancy, reproduction, dispersal and pathogenesis of *M. graminicola* and *S. tritici* is even poorer. However, results from other countries might be applicable in understanding this important pathogen. In Denmark, ascospores can serve as primary inoculum on emerging winter wheat crops in the autumn (Eriksen & Munk, 2003). Furthermore, the role of the teleomorph stage may go on during other periods of the growing season, as shown in the UK (Hunter *et al.*, 1999).

The disease patterns have changed during recent decades. As reported in the England and Wales survey, *S. tritici* became the most important leaf blotch disease during the mid 1980s (Hardwick *et al.*, 2001). Septoria tritici blotch is now the almost completely dominant foliar disease on winter wheat in Northern Ireland, but as in England and Wales the same shift or almost complete disappearance of stagonospora nodorum blotch by septoria tritici blotch happened in Northern Ireland (Mercer & Ruddock, 2004). Hypotheses put forward to explain why there was a shift from *S. nodorum* to *S. tritici* include *e.g.* less resistant varieties (Bayles, 1991), selection due to benzimidazole-resistance in *S. tritici* (Griffin & Fisher, 1985), and increased nitrogen fertilization during the period (Hardwick, 1998, Simón *et al.*, 2003). Bearchell *et al.*, (2005) found the ratio between *S. nodorum* and *M. graminicola* to be very strongly correlated with changes in SO_2 since 1843. In France, *S. nodorum* ceased to be an important parasite in the beginning of the 1990s and from then on the large majority of symptoms have been caused by *S. tritici* (Trottet, 2001).

Figure 8. Stagonospora nodorum blotch. Photo: Peder Waern.

Figure 9. Tan spot (Drechslera tritici repentis). Photo: Peder Waern.

Crop loss assessment

In the Kansas plant disease survey 1976-2000 the average yield loss due to the septoria tritici blotch and stagonospora nodorum blotch was 1.6%, but with a variation from very small yield losses in some years to very large losses in other years (Bockus *et al*., 2001). The mean yield loss due to septoria leaf blotch (including both stagonospora nodorum blotch and septoria tritici blotch) during the period 1970-1998 was 2.1% in England and Wales, within the range 0.1% to 8% (Figure 7). From the mid-1980s septoria tritici blotch was the most severe foliar disease of winter wheat in England and Wales (Polley & Thomas, 1991; Hardwick *et al*., 2001).

In Sweden, stagonospora nodorum blotch and septoria tritici blotch were among the most important diseases identified in field trials carried out 1978-1985 (Figure 8), later confirmed for a somewhat longer period (Andersson *et al*., 1986; Wiik *et al*., 1995). Andersson *et al*. (1986) indicated that the yield loss due to LBDs was even greater due to insufficient treatment effects. This was confirmed some years later by the use of the more effective strobilurins (Wiik *et al*., 1996; Bayles, 1999).

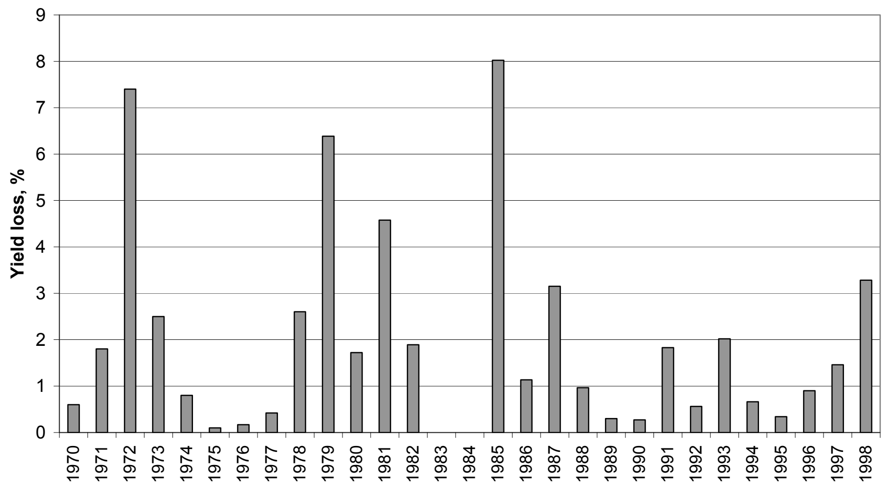

Figure 10. Estimated yield loss (%) due to *S. nodorum* and *S. tritici* in England and Wales during 1970-1998. Data compiled from King, 1977; Cook *et al*., 1991; Polley & Thomas, 1991; Hardwick *et al*. 2001. No values recorded 1983 and 1984.

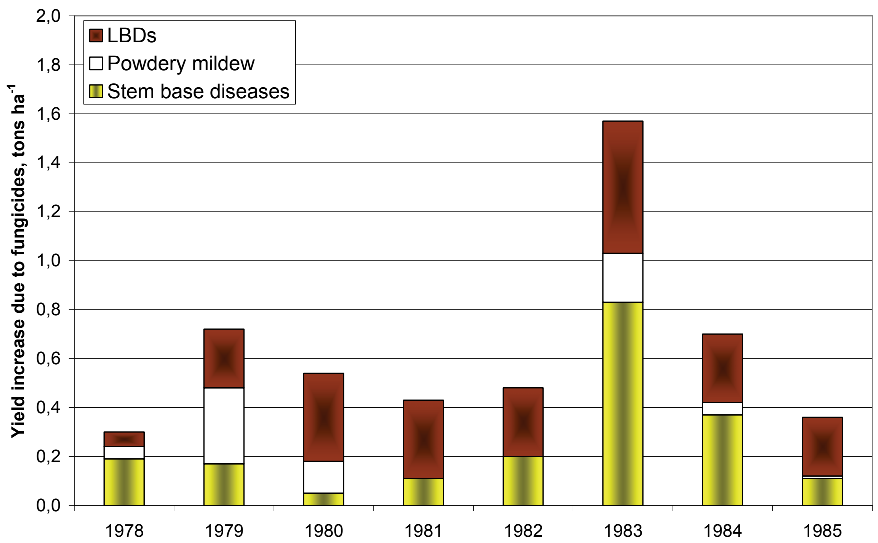

Figure 11. Estimated yield increase (tons ha^{-1}) due to effects of fungicide treatments on stem base diseases, powdery mildew and stagonospora nodorum blotch+septoria tritici blotch (LBDs) in 62 winter wheat field trials in southern Sweden 1978-1985 (Andersson *et al.*, 1986).

Disease cycle and epidemiology

In Indiana, USA, Shaner & Finney (1976) proposed a model with past and future precipitation and temperature to forecast very severe and severe epidemics of septoria tritici blotch. Bahat *et al.* (1980) found disease severity to be correlated with the number of dewy days and temperature index one to three weeks before formation of pycnidia. They also showed the role of the distance between consecutive leaves on the vertical progression of *S. tritici* pycnospores, *i.e.* the 'ladder' effect. Coakley *et al.* (1985) presented a similar model to Shaner & Finney (1976) but used consecutive days without precipitation instead of excess rain. In the equation proposed by Coakley *et al.* (1985), disease severity is explained by the consecutive days without precipitation between 26 March and 4 May (average heading 22 May) and minimum temperature ≤7 °C between 4 April and 3 May. In an experimental forecasting scheme. Shaw & Royle (1986) revealed two determining factors, the amount of *S. tritici* spores at GS 30 and precipitation and splash during GS 32-69. Spraying is recommended if there is raining four or more days with at least 1 mm rain during GS 39-55, or one day with at least 1 mm of rain during the last 14 days (Shaw & Royle, 1986). Shaw (1987) indicated that splash height was dependent on drop size range and not the volume of precipitation, a point confirmed by Walklate *et al.* (1989). Thomas *et al.* (1989) found that critical conditions for initial development of *S. tritici* occurred during early May, *i.e.* heavy rain giving at least 10 mm on one day or a total of 10 mm or more on two or three successive days, and they found the incubation period on the top three leaves to be 396-496 degree days. Daamen & Stol (1992) found incidence of *Septoria* spp. in May and severity in July to be positively correlated with cumulative precipitation over the months March-April and March-June, respectively. Furthermore, Daamen & Stol (1992) suggested that a sunny August will hamper the availability of ascospores. Temperature is important for infection by *M. graminicola,* and the rate of disease development and the ultimate disease severity (Magboul *et al.*, 1992). Shaw & Royle (1993) found the coincidence of rain splash and the emergence of the upper leaves to be of the utmost importance in forecasting severity of septoria tritici blotch, and that prevailing conditions almost always favoured infection. *Septoria* spp. treatment was recommended by Wiik (1993) when rainfall exceeds 30 mm or six days with ≥1 mm during 28 days before GS 55. Hansen *et al.* (1994) proposed a threshold for *Septoria* treatment to be seven or eight days with precipitation ≥1 mm during a 30-day period starting in GS 32. Smeets & Geypens (1995) looked for relationships between EPIPRE data from Belgium and Northern France and precipitation during two meteorological different seasons, 1992/1993 and 1993/1994. The scarcity of rain during tillering and stem elongation (February to May) in the 1992/1993 season and the abundance of rain in 1993/1994 gave an explosive spread of leaf blotch in the latter season (Smeets & Geypens, 1995). In the UK, severe infection with *S. tritici* occurred after heavy rain, 10 mm on one day or 10 mm on two or three consecutive days (Thomas *et al.*, 1989). In field experiments during 1988-1990, Cook *et al.* (1999) found clear indications that epidemics of foliar diseases initiated before flag leaf emergence had the greatest impact on yield. Parker *et al.* (1999) found negative correlations between temperature and disease severity, both temperatures below 7 °C and below -2 °C, the best model being provided by the latter temperature. Paveley *et al.* (2000) found leaf emergence of top leaves to be crucial for the timing of fungicides. In France, the rainfall and temperature in April and May are decisive for the development of *S. tritici* (Oste *et al.*, 2000). The IPM wheat model has been used in the Schleswig-Holstein region to identify whether fungicide treatments are worthwhile against important wheat diseases such as septoria tritici blotch (Verreet *et al.*, 2000). Weather criteria in this model include precipitation, relative humidity and leaf wetness. Verreet *et al.* (2000) found high precipitation, especially during

May, and little continuous frost in winter to promote the development of *S. tritici*. In a model developed from survey data on the two upper leaves during GS 73-75 in 1985-1996 in the UK, Gladders *et al.* (2001) found a range of risk variables to be important for septoria tritici blotch development. They found an increased number of frost days (≤2 °C) in November and high risk periods or rain splash events in May and June to increase the number of fields above a 5% severity threshold (Gladders *et al.*, 2001). They did not find any evidence supporting the indication by Daamen & Stol (1992) that sunshine duration decreased disease severity of *Septoria* spp. Temperatures below -2 °C during November and the first part of December have been found to be negatively correlated to severity of septoria tritici blotch (Parker *et al.*, 1999; Gladders *et al.*, 2001). Lovell *et al.* (2004) gave the causal mechanisms for this negative correlation, *i.e.* suppressed disease expression and a subsequent reduction of inoculum. In a two-step analysis, winter wheat fields with >5% disease severity were firstly identified with a qualitative model (epidemic or no epidemic) and then the severity of septoria tritici blotch was predicted with a quantitative model (Pietravalle *et al.*, 2003). In the qualitative model winter temperature during January/February and wind speed to about GS 31 were predictors, while in the quantitative model precipitation during tillering and stem elongation between April and middle May (GS 20-35) were predictors. Thus, the epidemic was first driven by temperature and subsequently by rain during stem elongation (Pietravalle *et al.*, 2003). Henze *et al.* (2007) found a relationship between temperature and the length of the latent period; a decrease of 0.2 day for an increase in the average temperature of 1 °C during the latent period. They found latent periods to range between 10 to 32 days, with an average of 20.4 ± 4.2 days. Furthermore, they found a decrease in the length of the latent period by 1.7 days per degree of northerly latitude in Northern Germany, but as temperature decreased northwards day length might be involved (Henze *et al.*, 2007). However, on a more global scale, Leath *et al.* (1993) found septoria tritici blotch to decrease with increasing distance from the equator, as opposed to stagonospora nodorum blotch, which increased. Shaw *et al.* (2008) concluded that weather factors previous to the growing season can influence annual variability of *M. graminicola*. This is supported by the hypothesis of Daamen & Stol (1992) that the frequency of ascospores decreased after a sunny August. Shaw *et al.* (2008) found no correlation between wind-run or the amount of wind in the spring and *M. graminicola* severity, as recorded by Pietravalle *et al.* (2003).

In the model by Zhang *et al.* (2007), most of the variability in the severity of septoria tritici blotch and other diseases was due to year effects. Furthermore, Gladders *et al.* (2001) found that year to year variation in disease severity was greater than variability at county level.

Fungicides and disease control

There are fungicides available to give good control against septoria tritici blotch (HGCA, 2009; SJV, 2009; Thygesen *et al.*, 2009; FRAC, 2009; www.pesticides.gov.uk, 2009). However fungicide resistance is an impending threat. During 2002, strobilurin treatments gave poor control against septoria tritici blotch in some areas, but sufficient control in most cases (Lucas, 2003). However, during 2003 strobilurin resistance in *S. tritici* was widespread and later investigations showed that resistance emerged independently at several locations (Fraaije *et al.*, 2005; Torriani *et al.*, 2009). Some of the azoles will hopefully continue to offer robust control but an alarming decrease in sensitivity has been reported (Mavroeidi & Shaw, 2005; Leroux *et al.*, 2007; Brunner *et al.*, 2008).

Different kinds of forecasting and warning systems have been proposed to prevent unnecessary use of fungicides and to optimise fungicide input. Some systems are simple, others complex, some are under development, some in use and some not in use (Reinink, 1986; Emmerman *et al.*, 1988; Verreet, 1995; Schepers *et al.*, 1996; Jørgensen *et al.*, 1999; Verreet *et al.*, 2000; DESSAC, 2000; Milne *et al.*, 2007; www.cropmonitor.co.uk, 2009). Six decision support systems for fungicide input against septoria tritici blotch in winter wheat were tested in Ireland during 2003-2005 by Burke & Dunne (2008). The in-crop Septoria Timer (Thies CLIMA, www.thiesclima.com, 2009) responding to both precipitation and simulated leaf wetness reduced disease severity on leaves F-2 and F-3 on both Madrigal (susceptible) and Claire (moderately resistant) in two out of three years and gave a the higher margin over fungicide cost than five interactive DSSs and standard 2-spray and 3-spray treatments. Many of these forecasting systems demand a great deal of work, but the gain is the integration of control measures in a straightforward and lucid system, and profitable use of fungicides.

2.5.2 Powdery mildew and the rusts

Powdery mildew and the rusts are described together here due to similarities in some aspects important for this thesis. Of course the pathogens differ in many aspects, *e.g. Blumeria graminis* belongs to the powdery mildews, a vast group of nearly 10 000 species attacking angiosperms, to which *B. graminis* f.sp. *tritici* is evidently more related than to the rusts, and within the three wheat rust species – *P. graminis, P. striiformis* and *P. triticina* – there are quite large differences (Hau & de Vallavieille-Pope, 1998; Glawe, 2008).

Figure 12. Powdery mildew. Photo: Peder Waern.

The pathogens

Both powdery mildew and the rusts are parasitic biotrophs, meaning that they obtain their nutrients from living host tissue. These pathogens are 'shifty enemies' due to their high ability for adaptation (McDonald & Linde, 2002; Stakman, 1948 *cit.* in Kolmer *et al.*, 2007).

B. graminis (syn. *Erysiphe graminis*) is the only species out of 650 in the family *Erysiphaceae* causing powdery mildew of cereals, and *B. graminis* f.sp. *tritici* is the *formae speciales* specialized on wheat. Differences are now being explored in molecular phylogeny and results reveal that the order *Erysiphales* belongs to *Leotiomycetes* instead of *Pyrenomycetes* and isolates from *Agropyron*, *Secale* and *Triticum* belong to one out of nine distinct groups of different *formae speciales* of *B. graminis* (Inuma *et al.*, 2007; Glawe, 2008). Today about 60 resistance alleles located at 37 loci have been identified in the wheat genome, and along with the gene-for-gene concept several specific races of *B. graminis* are known (www.usda.ars.gov, 2009).

Figure 13. Brown rust. Photo: Peder Waern.

Figure 14. Yellow rust. Photo: Peder Waern.

The names of the three rust cereal diseases can be confusing due to different common names in English and American, *i.e.* yellow rust and stripe rust, brown rust and leaf rust, black stem rust and stem rust, respectively. The English names concur with Swedish usage and are used here. Black stem rust (*P. graminis* f.sp. *tritici*) is a very rare pathogen in Sweden nowdays. With the exception of one year (1951), black stem rust was unimportant in the 20th century and is therefore not considered further in this thesis, but it should not be overlooked as it is an apparent potential disease (Roelfs, 1989).

Yellow rust has been reported in more than 60 countries and found on all continents except Antarctica (Chen, 2005). Contrary to black stem rust, no alternate host is known for yellow rust and accordingly no pycnial or aecial stages occur. A few *formae speciales* are known, including hosts such as wheat, barley, rye and a few other grasses. DNA methodology can now clarify the relationship between yellow rusts from different hosts and the different *formae speciales* no longer have to be argued (Chen *et al.*, 1995). Today 70 genes for resistance against yellow rust are known. In the USA 121 races of yellow rust on wheat caused by *P. striiformis* f. sp. *tritici* were detected recently, half of these since the year 2000. Four new differentials (a differential is a line or a cultivar used to differentiate and to determine races of the fungus) added during 2000 contributed to this recent increase in races (Chen, 2007).

Brown rust is the most common and widely distributed disease of wheat in the USA and probably worldwide (Kolmer *et al.*, 2007). According to Kolmer (1991), no susceptible alternate hosts have been found in the USA, and the Central Disease Laboratory (http://www.ars.usda.gov/mwa/cdl, 2009) states that 'sexual aeciespores do not start epidemics'. More than 50 resistance genes against brown rust have been described, most of them race-specific (Kolmer *et al.*, 2006). Every year, 40 to 60 races are characterised in the USA and 40 races (virulence phenotypes) in Canada on near-isogenic lines of the variety Thatcher (McCallum & Seto-Goh, 2008).

Crop loss assessment

In England, almost 50 years ago the percentage yield loss of grain due to powdery mildew was estimated to be $2\sqrt{x}$, where x is the percentage of mildew at GS 58-59, and losses over all experiments were slightly more than 0.12 tons ha^{-1} or about 1 cwt per acre (Large & Doling, 1963). However, powdery mildew ought to be assessed all through the season to get the best relationship to crop growth, yield and grain quality (Jenkyn & Bainbridge, 1978). In addition, the usual situation in the field is the presence of multiple disorders, which increases the complexity, *e.g.* when mildew and snow mould are considered together (Richardson *et al.*, 1976). In nine field trials in southern Sweden 1979-1980, yield loss due to powdery mildew was estimated to be 4-10%, but the attacks were quite small and effects of Bayleton 25 WP (a.i. triadimefon) other fungal pathogens could not be excluded (Wiik, 1981, 1982). Leath & Bowen (1989) recorded a yield reduction of 17% when mildew severity on the flag leaf reached 10% at heading. In Kansas during 1976-1987, the mean disease loss estimate due to powdery mildew was 0.6% with an annual range of 0.1-1.3% (Sim IV *et al.* 1988). In field trials with yield levels of 7-9 tons ha^{-1} in the Netherlands, Daamen (1989) estimated a damage function with mildew intensity over time to be -0.0013 tons ha^{-1} per pustule-day of mildew per leaf, from the second node stage to early dough stage. In England and Wales mean annual yield loss due to powdery mildew was substantial during the first part of the period 1976-1998 but during the latter part of this period it decreased considerably (Figure 9) (King, 1977; Cook *et al.*, 1991; Polley & Thomas, 1991; Hardwick *et al.*, 2001).

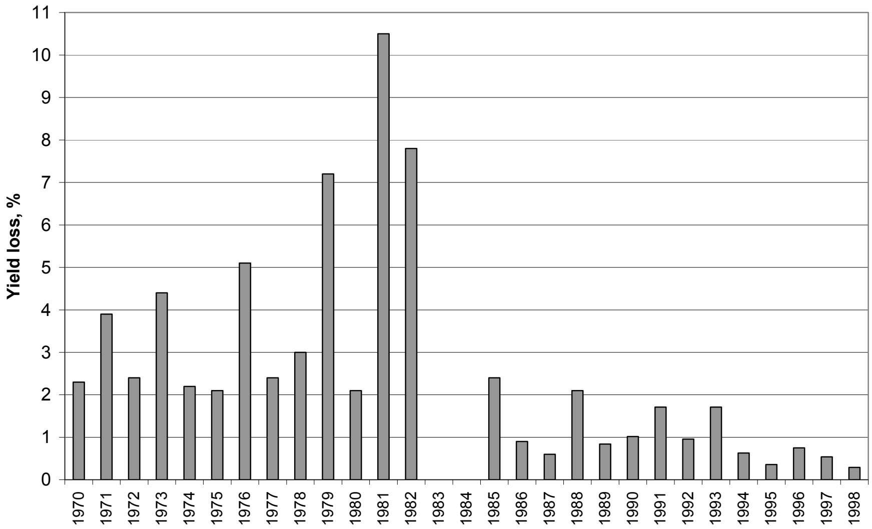

Figure 15. Estimated yield loss (%) due to powdery mildew in England and Wales 1970-1998. Compiled from King, 1977; Cook *et al.*, 1991; Polley & Thomas, 1991; Hardwick *et al.*, 2001.

In the USA, crop losses due to the rusts in cereals have long been estimated (http://www.ars.usda.gov/mwa/cdl). Figure 10 presents annual mean crop losses for the period 1976-2008. The mean loss due to brown rust was almost three times larger than the mean loss due to yellow rust during these years, 2.2% and 0.8%, respectively. Each percentage unit of yield loss corresponded to about 25 kg grain ha^{-1}. In 28 out of 33 years, yield loss due to brown rust exceeded that of yellow rust. Yield loss due to yellow rust exceeded that due to brown rust in four years, especially recent years during (2001, 2003 and 2005). As can be seen in Figure 10, national mean yield losses differ substantially over years but also between states (not shown) for each of the rusts in the USA. In England and Wales during 1976-1998, mean yield loss due to brown rust and yellow rust was quite small in comparison with that in the USA, 0.05% and 0.07% respectively (King, 1977; Cook *et al.*, 1991; Polley & Thomas, 1991; Hardwick *et al.*, 2001). Thus, yield loss due to a particular disease can differ between countries, in time and space due to the access to resistant varieties and to the farmers' choice of varieties, the environment and other factors.

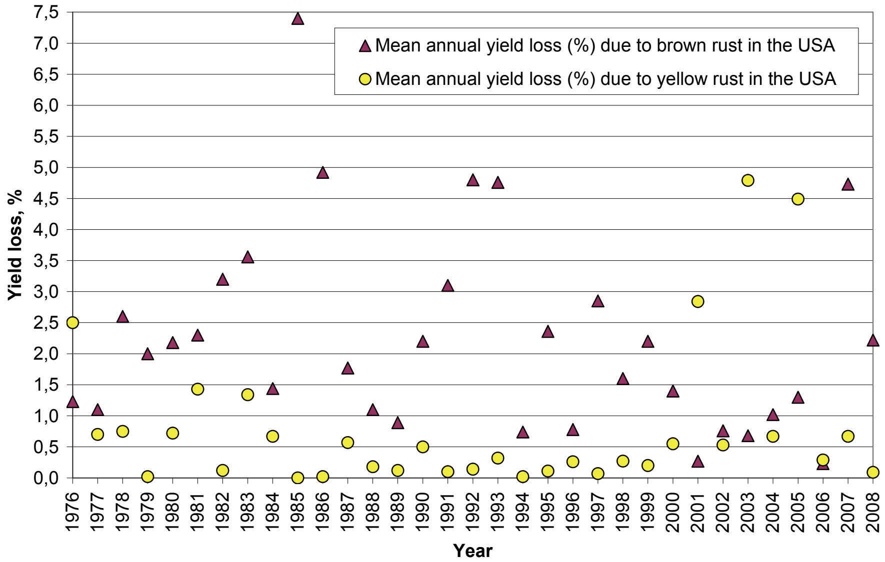

Figure 16. Annual yield loss due to brown rust and yellow rust in the USA 1976-2008. Compiled from data on the website of the Cereal Disease Laboratory, University of Minnesota, St. Paul (http://www.ars.usda.gov/mwa/cdl).

In Sweden annual surveys are carried out but these are not coupled to yield loss equations. On the other hand, yield losses are considered in field trials. A review of crop losses due to winter wheat diseases in field trials in southern Sweden 1978-1985 concluded that either brown rust or yellow rust contributed to the yield loss (Andersson *et al.* 1986). However, some years later, during the end of the 1980s and beginning of 1990s, winter wheat in both southern and central Sweden was attacked more than usual by brown rust due to mild winters, establishment of the rust already in the autumn and probably incoming spores from neighbouring countries (Waern, 1989; Wiik, 1990; Wiik *et al.* 1995). In field trials in Sweden with untreated and fungicide-treated plots during these years, the relationship between yield (Y, tons ha^{-1}) and brown rust (x, within the range 13-66% severity at GS 83-87) was estimated to be (Wiik, 1990):

$$Y=8.85-19x,\ R^2=0.93 \qquad \text{(equation 1)}$$

During recent times, brown rust has been reported to give the same high yield losses in some field trials as with equation 1, *i.e.* yield losses of around 1.0-1.5 tons ha^{-1} corresponding to 10-15% (Ewaldz & Berg, 2008).

In 2008, severe attacks of yellow rust were reported in a Dutch variety (Tulsa) in southern Sweden (Ewaldz & Berg, 2009). Different fungicide treatments resulted in 2.0 to 2.9 tons ha^{-1} yield increase in one field trial, corresponding to 20-30% yield loss. With a down-graded Swedish plant breeding programme and a market based on varieties not bred in Sweden, it will be increasingly important in the future to be prepared for unexpected plant diseases. If we are not prepared, it might be difficult to control them and avoid yield losses.

Disease cycle and epidemiology

Powdery mildew and the rusts have many epidemiological similarities. They are both polycyclic diseases with several infection cycles each season. Epidemiologically, powdery mildew and the rusts can be considered to be r-strategists, *i.e.* fast disease development, long distance dispersal and rather frequent epiphytotics arising in favourable weather on susceptible varieties and on varieties that were previously race-specific resistant.

According to Hermansen *et al.* (1978), spores of powdery mildew and rust can travel by the wind many hundreds of kilometres from the British Isles to Jutland in Denmark. Long distance dispersal by means of direct movement of airborne spores (500 km or more) or by human-aided means, *e.g.* on wheat straw used for animal bedding during transport or attached to air passengers is common (Hermansen *et al.*, 1978; Brown & Hovmøller, 2002; Wellings, 2007). Recently the ancestry of the powdery mildew population in the USA has been explored using DNA markers. Long-distance dispersal from Europe seems more likely than ancestry for mildew on wild native grass hosts in the USA (Parks *et al.*, 2009).

Tapke (1951) referred to several older investigations giving examples of many types of contradictory climates that promote mildew, but stressed the importance of the environmental conditions to which plants are exposed before inoculation. Aust & v. Hoyningen-Huene (1986) proposed cooperation in holistic field experiments between phytopathologists, agrometeorologists and physiologists to reveal the complicated interactions between microclimate, powdery mildew and plant. Daamen *et al.* (1992) found that percentage prevalence of mildew in fields in May was correlated to temperature in the months October, December, January, February and March. Friedrich (1995) described the effects of temperature, air humidity, wind speed and precipitation on different stages in the disease cycle, *e.g.* on spore dispersal and found several factors to have a negative effect on the infection probability, such as precipitation, the duration of precipitation, high wind velocity, and very low and very high vapour pressure deficits. Rossi & Giosuè (2003) found good agreement between actual and predicted powdery mildew severity in Italy by using temperature, vapour pressure deficit, precipitation and wind from standard meteorological stations in a dynamic model to calculate times of incubation, latency and sporulation periods. Te Beest *et al.* (2008) found temperature, humidity and rain in April to June to best explain disease severity, and wind to predict a damaging epidemic (>5 % severity).

In comprehensive reviews by Line (2002) and Chen (2005) yellow rust is described as very dependent on weather conditions, specifically moisture, temperature and wind. Coakley and co-workers published several papers during the 1980s on models for predicting yellow rust on winter wheat in the Pacific Northwest of the USA, and by using an improved method (the Window Pane program) found temperature and spring precipitation to be influential factors (Coakley *et al.*, 1988; Coakley, 1988). They also found high temperature (>25°C) to be important in promoting adult plant resistance. Daamen *et al.* (1992) indicated that winter survival of yellow rust at high temperatures during November-March was the critical factor, and not summer survival with rain during the previous summer and autumn. Vechet (1992) found that precipitation and temperature were most important factors during the beginning of the epidemic, *i.e.* the first incubation period. Gladders *et al.* (2007), van den Berg & van den Bosch (2007) and Te Beest *et al.* (2008) emphasized temperature as the most important weather variable, and its effects on reproduction and winter survival. Papastamati & van den Bosch (2007) also emphasized temperature for yellow rust progress and added dew period and light quantity.

Clifford & Harris (1981) claimed that brown rust was a potential disease to winter wheat in Britain. The range of temperature, moisture and light intensities were suitable under UK conditions for several stages in the disease cycle of brown rust. However, formation of infection structures needs high temperature which might explain why the disease occurred too late in the season to be a substantial problem. In southwest France, unfavourable temperature and free moisture seem to be the weather factors that delay brown rust development (Benizri & Projetti, 1992). Daamen *et al.* (1992) in the Netherlands found brown rust to be favoured by high temperature in March and cumulative precipitation in April and May. The high temperature in March probably facilitated sporulation and infection processes on young leaves. In Italy, brown rust intensity was found to vary from year to year due to weather conditions and a model used to simulate this variation included the variables temperature, relative humidity, and precipitation, and estimated daily mean, minimum and maximum temperature and leaf wetness duration (Rossi *et al.*, 1997). Eversmeyer & Kramer (1998) described the importance of moisture and temperature for brown rust establishment on volunteer plants making the green-bridge phenomena possible in the Great Plains, USA. Dew formation caused by temperature shifts supported infection on both volunteer plants and on the next early-sown crop, while high temperature, more moist conditions than normal and snow cover made survival of brown rust possible. In Argentina, brown rust severity was adequately predicted using a model including daily mean temperature >12 °C and days with relative humidity >70% without precipitation and a cultivar resistance index (Moschinin & Pérez, 1999).

Fungicides and disease control

Effective fungicides against powdery mildew and the rusts have been used for decades (Jenkins & Lescar, 1980; Hollomon & Wheeler, 2002). Seed dressing agents such as oxycarboxin and triadimenol can prevent early attacks of powdery mildew and rusts. However, more durable control can be achieved by controlling the diseases during the growing season. The use of fungicides applied to growing winter wheat began to increase notably during the 1970s and 1980s, when rust and powdery mildew could be effectively controlled by the systemic fungicide Bayleton (a.i. triadimefon). In the USA, the first commercial use of fungicides carried out against a yellow rust epiphytotic in 1981, prevented severe yield loss at a multimillion dollar level (Line, 2002). Since then new sterol biosynthesis inhibitors, strobilurins and other active ingredients have been used worldwide (Bartlett *et al.*, 2002). Unfortunately, fungicide resistance has been quite a problem for many systemic fungicides, for example wheat powdery mildew, snow mold, septoria tritici blotch and tan spot are now resistant to strobilurins (FRAC, 2009). By a single point mutation one amino acid changed position which led to strobilurin resistance in wheat powdery mildew (Sierotzki *et al.*, 2000). However, effective fungicides against powdery mildew and rusts are still available and the development of new fungicides continues, governed by official legislation and regulations. In Sweden, products with the active ingredients fenpropidin and metrafenon have good efficacy against powdery mildew, and the strobilurin pyraclostrobin is in the forefront against yellow rust and brown rust (SJV, 2009).

The integrated disease management programme MoreCrop (Managerial Options for Reasonable Economical Control of Rusts and Other Pathogens) using the concept of the disease triangle, was developed during the 1990s in the USA. In MoreCrop, information and recommendations on disease control using methods including fungicides and resistant cultivars are based on more than 40 years of experience (Cu & Line, 1994; http://pnw-ag.wsu.edu/MoreCrop.htm, 2009).

2.5.3 Eyespot

The pathogens

Eyespot (syn. strawbreaker foot rot) was long considered to be caused by one fungus, *Pseudocercosporella herpotrichoides*, but divided into two main pathotypes, the W-type and the R-type, or two varieties, *P. herpotrichoides* var. *herpotrichoides* and *P. herpotrichoides* var. *acuformis*.

Figure 17. Eyespot. Photo: Peder Waern.

The W-type (wheat-type) is more pathogenic to wheat than to barley and rye, while the R-type (rye-type) is equally pathogenic to wheat, barley and rye. The disease complex of eyespot became even more complicated as two more pathotypes were recognized – the C- and S-types, also pathogenic to wild grass hosts. Furthermore, two more species seemed to be involved, *P. anguioides* and *P. aestiva* (Fitt *et al*., 1988: Lucas *et al*., 2000). The sexual stages of the eyespot fungi were detected during the 1980s and 1990s. Crous *et al*. (2003) proposed *Oculimacula* for the teleomorphs or the sexual form. *O. yallundae* (previously the W-type) and *O. acuformis* (previously the R-type) are now cited in recent publications as the taxa that causes eyespot (Chapman *et al*., 2008; Leonard *et al*., 2008). Selection forces such as the use of fungicides, act on the balance between the eyespot fungi. An example is the change in population proportions like that during 2004 in the UK from *O. acuformis*, the most common until that time, to *O. yallundae*. Such changes might influence fungicide efficacy (Bateman, 2002; Burnett, 2005; Fitt *et al*., 2006). The host range of the eyespot fungi is large and includes wheat, rye, barley, oats and many wild and cultivated grass hosts (Lucas *et al*., 2000). Sharp eyespot, another stem-base disease caused by *Rhicoctonia cerealis* damages the stem-base in a similar way to eyespot. As the name indicates the eyespot lesions of sharp eyespot are more sharply delineated than those caused by *Oculimacula* spp.

Crop loss assessment

In surveys in England and Wales, yield losses due to eyespot were estimated to be 0.3 to 1.2% during 1975-1980, and in field trials yield loss was slightly more than 10% in the absence of lodging and almost 20% with lodging (Scott & Hollins, 1974; Clarkson, 1981). Jones (1994) recorded mean yield increase after fungicide treatment in the range 0.36 to 0.85 tons ha^{-1} in 40 field trials carried out in the UK 1983-1986. Typical average yield increases due to treatment with benzimidazole and prochloraz in Sweden were only slightly more than 0.2 and 0.3 tons ha^{-1}, respectively (Wiik *et al.*, 1995). Evaluations of the relationship between eyespot and yield increase due to fungicide treatment have been carried out in a few countries, e.g. in Sweden one evaluation for 1975-1989 included 82 field trials with benzimidazole while another 90 field trials in 1987-2001 included azoles and pyrimidines (Olvång, 1990; Wiik, unpubl.). The results from these evaluations show a large variation and typical relationships between Y (yield increase due to spraying against eyespot at GS ~31) and x (an eyespot index where 0 is no attack, 10-20 low attacks, >30 severe attacks) are presented in equations 2 (Olvång, 1990) and 3 (Wiik, unpubl.) using estimations given in Table 2:

$$Y = 167–2.45x+0.25x^2,\ R^2=0.31 \qquad \text{(equation 2).}$$
$$Y = 204–19x+0.77x^2,\ R^2=0.42, \qquad \text{(equation 3)}$$

Table 2. *Estimated yield increase (tons ha^{-1}) due to fungicide treatment against eyespot at different levels of eyespot attack (eyespot index 0 is no attack, 10-20 low attacks, >30 severe attacks)*

Eyespot index in untreated plots	Estimated yield increase (tons ha^{-1}) according to equation 2	Estimated yield increase (tons ha^{-1}) according to equation 3
0	0.17	0.20
10	0.17	0.09
20	0.22	0.13
30	0.32	0.33
40	0.47	0.68
50	0.67	1.18

Quite a strong correlation was found between lodging and yield increase due to fungicide treatment against eyespot which reveals the importance of treatment against eyespot attack when leading to lodging, although such attacks occur very rarely (Wiik, 1986). However, the correlation between eyespot and yield increase due to fungicide treatment against eyespot was very weak. In contrast, Burnett & Hughes (2004) found a significant association between eyespot levels and yield, but a weaker correlation between lodging and yield loss. The difference in the relationship between eyespot and yield is probably due to uncertain results from the visual assessments made by Wiik (1986) and the more certain PCR methodology used by Burnett & Hughes (2004). Wiik (1986) indicated that fungi other than eyespot were probably present on the stem bases, *e.g. S. nodorum* and *Fusarium* spp.

Disease cycle and epidemiology

The eyespot fungi are favoured in cool and temperate regions such as north-western Europe, the Pacific Northwest in the USA and New Zealand. Under wet conditions, conidia are produced on the straw stubble or infected debris and dispersed by rain splash within a field over short distances throughout October to July, but especially during March and April (Hollins & Scott, 1980; Fitt *et al.*, 1988). Airborne ascospores from apothecia can be spread over longer distances and pose a threat to first wheats (Burnett & Hughes, 2004). Fitt *et al.* (1988) described four important stages in eyepot attacks:

- establishment of the eyespot fungi on leaf sheaths,
- penetration of successive leaf sheaths,
- establishment of the eyespot fungi on stems,
- stem lesion enlargement.

The eyespot symptoms seen on the stems late in the season are the outcome of fungal progress through all the succesive leaf sheaths, ending up with attacks on the stem. Severe attacks of eyespot weaken the stem and cause eyespot-induced lodging (Scott & Hollins, 1978). Fitt *et al.* (1988) concluded that conditions are usually favourable for the establishment on leaf sheaths in the UK. All four stages are favoured by weather factors. A crucial stage in stem lesion establishment seems to be when rapid death of leaf sheaths prevents attacks on the stem. In German investigations during the 1960s and 1970s, temperatures between 4 and 13 °C for at least 15 h and relative humidity > 80% were found to be most favourable for infection (*cit.* Fitt *et al.*, 1988).

Fungicides and disease control

Fungicides with good efficacy against eyespot are available today, but fungicide resistance has excluded the continued use of benzimidazoles since the beginning of the 1980s in many European countries and later in the Pacific Northwest of the USA, and in the beginning of the 1990s the DMI (dimethylation inhibitor) prochloraz in France (King & Griffin, 1985; Murray, 1996; Leroux & Gredt, 1997). Cyprodinil seems to be the best fungicide available for the moment (Ray *et al.*, 2004; Burnett, 2005). However, severe attacks of eyespot are rare, especially those leading to lodging and significant yield loss. Good straw strength in varieties limits the damage due to eyespot but agricultural practices may promote eyespot attacks. It is not easy to predict whether a fungicide treatment against eyespot will be profitable because several factors have an impact on the outcome of treatment. Fitt *et al.* (1988) did not believe a pure weather-based forecasting system to be appropriate in the UK. A risk assessment method by means of a risk algorithm was developed and validated. By definition, the risk algorithm used a fixed set of factors including previous crop, tillage, eyespot at GS 31-32, sowing date, precipitation in March and April and soil type and weighed them after importance. The conclusion of the project was that the risk algorithm has to be refined, especially regarding weather factors and probabilistic weather modelling and that the results of the existing risk assessment method should be used as guidelines rather than as strict rules on when to spray (Burnett & Hughes, 2004; Burnett, 2005).

3 Aims and objectives

The overall aims of this thesis were to identify and quantify biotic and abiotic yield constraints in winter wheat in southern Sweden and to identify relationships between winter wheat yield and these biotic and abiotic yield constraints. Specific objectives were to provide useful data for forecasts and warnings on plant diseases of winter wheat, thereby optimising fungicide input. Biotic constraints in this study were limited to plant diseases that can be controlled with fungicides, *i.e.* the Leaf Blotch Diseases [LBDs, including septoria tritici blotch caused by *Mycosphaerella graminicola* (anamorph *Septoria tritici* – the major leaf blotch disease in Sweden), stagonospora nodorum blotch caused by *Phaeosphaeria nodorum* (anamorph *Stagonospora nodorum*), tan spot caused by *Pyrenophora tritici-repentis* (anamorph *Drechslera tritici-repentis*)], powdery mildew caused by *Blumeria graminis*, brown rust caused by *Puccinia triticina*, yellow rust caused by *Puccinia striiformis* and eyespot caused by *Oculimacula acuformis* and *Oculimacula yallundae*. The abiotic constraints in this study were limited to those measured in the field trials and nearby temperature and precipitation measurements.

In Paper I, results from field trials in winter wheat carried out during recent decades in southern Sweden were evaluated with emphasis on yield gain due to fungicidal control of diseases. The objectives were to examine the relationships between fungicide treatments and yield and multiple diseases and yield, and to determine variations in yields and diseases within and between years.

In Paper II, long-term relationships between yield of winter wheat and temperature/precipitation and between plant disease attack and temperature/precipitation were evaluated. In addition, the use of disease severity and disease incidence as predictors was compared and the potential use of weather factors in plant disease prediction was evaluated.

In Paper III, the profitability of fungicide use in field trials carried out in southern Sweden was evaluated in a more thorough economic analysis than usual, supplemented with scenarios with different grain prices and fungicide treatment costs expected to be relevant in future assessments. The aim of the evaluation was to highlight economic considerations in wheat production and to examine the profitability of a single fungicide treatment at GS 45-61 in winter wheat.

In Paper IV, results on diseases and yield of winter wheat from field trials in southern Sweden were evaluated in relation to soil factors, agricultural practices and management. The objectives were to find relationships between yield and soil factors, agricultural practices including timing and dates of events, as well as the relationships between plant diseases and soil factors, agricultural practices including timing and dates of events.

4 Materials and methods

4.1 Field experiments (Papers I-IV)

Field experiments were carried out in southern Sweden (Scania), a pronounced agricultural area of Sweden where about 30% of the approx. 300 000 ha of winter wheat usually grown in Sweden are located. Data were obtained from more than 400 field trials in winter wheat fields during the period 1977-2007. The field trials were carried out on farms using different cultivars and agricultural practices, *e.g.* fertiliser doses. All interventions except fungicide treatment were usually carried out by the farmers. However, in 20% of the field trials three levels of nitrogen and up to seven varieties were included in the same field trial. The field trials comprised four replicates in randomised blocks but in the evaluations mean results from each field trial were used. The field trials were carried out by staff at the Rural Economy and Agricultural Societies according to a precise protocol. Untreated and treated refer to plots in the field trials not treated with fungicides opposed to those plots that were treated. Growth stages (GS) according to Tottman & Broad (1987) were used: In addition to an untreated control, one or more of the following four treatments were included in Paper I: 1) A single early treatment at GS 30-33 (mean GS 31 May 17) with fungicides effective primarily against eyespot, 1977-2002; 2) a single treatment with fungicides against LBDs, mildew, yellow rust and brown rust just before/during heading at GS 45-61 (mean GS 53 June 14), 1983-2005; 3) a split treatment with fungicides against leaf diseases including an early treatment at GS 31-40 followed by one at GS 45-61 and 4) a split treatment with an early treatment at GS 30-33 primarily against eyespot, followed by a treatment at GS 45-61 with fungicides against leaf diseases. In early years, eyespot was primarily treated with benzimidazoles and LBDs with azoles, but also amides and a conazole-morpholine mixture. In later years, pyrimidines were generally used against eyespot and strobilurins, conazoles and morpholines against leaf diseases, often in different combinations. Only a single treatment was carried out at GS 45-61. In Papers II, III and IV one single fungicide treatment in winter wheat was used during a limited growth period (GS 45-61) shown to hold the most important GS for LBDs in Sweden (Wiik *et al.*, 1995; Paper I). Standard products at recommended dosages were generally used, *e.g.* 0.5 L ha^{-1} Tilt 250 EC, a.i. propiconazole 250 g L^{-1}; 0.8-1.0 L ha^{-1} Tilt Top 500 EC, a.i. propiconazole 125 g L^{-1} + fenpropimorph 375 g L^{-1}; 0.5-1.0 L ha^{-1} Amistar, a.i. azoxystrobin 250 g L^{-1}.

Yields from usually 20 m^2 per plot were harvested and yield quality attributes included yield and thousand grain weight reported at 15% water content, hectolitre weight, kernel protein and Hagberg falling number. Soil factors from a soil sample taken from each field trial site at sowing included analyses of organic matter, clay content, sand and silt fraction and the amount of soluble phosphorus, potassium, magnesium and calcium by the AL-method with ammonium lactate. For each field trial the nitrogen dose was recorded (comprising nitrogen from the previous crop, manure and mineral nitrogen by fertilisation). Timing factors recorded were day of sowing, Julian day at GS 55, Julian day at spraying and Julian day at harvest and number of days from sowing to harvest. Disease severity variables included records of percentage damage to flag leaf (F) and leaf 2 (F-1) at GS 75 and to leaf 3 (F-2) at GS 55 for LBDs, and maximum assessed attacks on any of the upper leaves of powdery mildew, yellow rust, brown rust, and disease index for eyespot. Data were collected on site factors and agricultural practices in the field trials. Previous crops were distributed into six classes. Of a total of 25 varieties at different trial locations, only a few such as Ritmo, Kris, Kosack, Bill, Marshal and Folke were used in more than 39 up to 144 field trials each. Mineral nitrogen, Julian day of sowing and Julian day when the crop reached GS 55 were distributed into five classes each. Furthermore, the data were distributed into two counties, seven regions and three fungicide types. Missing data on some variables were quite common, *e.g.* protein content and Hagberg falling number.

4.2 Surveys (Paper II)

Disease surveys and forecasts were carried out in wheat fields in southern Sweden (Scania) during the period 1988-2007. Each field contained a marked plot in which no treatment with fungicides or insecticides was allowed but the plot was treated similarly to the rest of the field in every other respect. Crop growth stage (GS) and disease incidence of LBDs (assessed collectively), powdery mildew, yellow rust and brown rust were recorded every week from late April to early July, ~GS 24 to ~GS 75. An eyespot index was calculated from assessments on samples taken in July at ~GS 75. Plant disease severity from field trials was compared with plant disease incidence from surveys.

4.3 Meteorological data (Paper II)

Temperature and precipitation data were obtained from the Swedish Meteorological and Hydrological Institute (SMHI, 2009). Weather stations in the main agricultural areas in southern Sweden (Scania) were chosen, *i.e.* the same areas in which the field trials and surveys were carried out. Temperature and precipitation data from 36 stations were pooled to produce a mean value for each month and year for southern Sweden during 1983–2007.

4.4 Economics (Paper III)

Cost calculations were based on data from Agriwise (2009), the Swedish Rural Economy and Agricultural Societies and the Swedish Board of Agriculture (SJV, 2009) and adjusted estimations of damage owing to fungicide application reported by Folkesson (1992). All calculations were made in Swedish crowns (SEK) and converted to euro (€) at an exchange rate of 10 SEK to 1 €.

4.5 Site factors and agricultural practices (Paper IV)

Site factors, soil factors, nitrogen and timing factors see 4.1.

4.6 Statistics

Conventional statistical analyses performed by the well-known programmes SPSS and SAS were used. For details see Papers I–IV.

5 Results and discussion

The long-term field trials evaluated in this thesis demonstrated large variations within and between years and changes occurring over years during the period 1977-2007. The aim of the thesis was to explain these variations and changes which can be attributed to many factors, not least to the impact of man.

In the introduction, the disease tetrahedron was used to demonstrate interactions between the components of plant disease epiphytotics. This is a good starting point for this thesis. In Paper I the interactions or relationships between the host winter wheat and some of the fungal pathogens causing diseases in this host are described. The interrelationships between the environment (in this case by temperature and precipitation), winter wheat and fungal pathogens are described in Paper II. The economics of yield gains due to fungicide treatment are described in Paper III. The relationships between site factors, agricultural practices, yield and fungal pathogens are described in Paper IV.

5.1 Grain yield quantity

The green revolution during the 1960s, when grain yield of wheat in Asia was multiplied several-fold in a short period of time, also occured in other parts of the world, although over a longer period. For example in Sweden, grain yield has tripled since the 1940s and doubled since the 1960s. The dramatic increases in Asia and in other parts of the world are due to the use of fertilizers and pesticides, technical innovations and progress in plant breeding. The debate on how this exploitation of resources will influence the prospects of future generations continues.

In the present study, a single eyespot treatment improved yield by ~320 kg ha^{-1} yr^{-1} during the period 1977-2002, mainly due to occasional years with severe eyespot (Paper I). The yield increase due to a single fungicide treatment at GS 45-61 increased yield by 9.9% or 660 kg ha^{-1} yr^{-1} for 1983-1994 and 10.7% or 970 kg ha^{-1} yr^{-1} for 1995-2005. An additional, extra-early treatment at GS 30-40 against LBDs increased yield by ~250 kg ha^{-1} yr^{-1}. Yield of both untreated and fungicide-treated plots increased from approx. 6000 to 12000 kg ha^{-1} over the period 1983-2005. The yield increase due to fungicide treatment did not markedly continue to grow in the field trials during 1983-2007, in spite of higher yield levels and the introduction of more effective fungicides. To be exact, there was a slightly higher annual increase over time in fungicide-treated plots compared with untreated, 217 kg ha^{-1} and 203 kg ha^{-1} (Paper I), but not more, and the regression lines of the annual mean yields relative to the mean yield 1983-2007 in untreated plots and fungicide-treated are parallel (Figure 11). This tells us that factors other than fungicides have contributed to the continuously increasing yield during this period. These factors may include the continual introduction of more high-yielding varieties (varieties with improved lodging resistance, higher harvest index, more grains per unit area, earlier anthesis, longer grain filling period, higher N use efficiency and disease resistance), and also improved sowing techniques, better capacity and agricultural practices, factors not assessed in this study. In addition, changes in climate, the performance of field trials and the extension of knowledge during these 25 years are other factors to be considered (Austin, 1999; Brancourt-Hulmel *et al.*, 2003).

Lovell *et al.* (1997) and Ewaldz (2000) reported the yield increase due to fungicide treatment to be smaller at higher yield levels and attributed this to less vertical upward movement of *S. tritici* inoculum in a dense canopy than in a thin canopy. This finding is in agreement with results reported in this thesis, *i.e.* that LBD severity on both leaf 3 at GS 55 and leaf 2 at GS 75 was negatively correlated with yield level. However, in the results reported here, the absolute yield increase due to fungicide treatment, increased more at the higher of two yield levels, but the percentage increase was lower (Paper I).

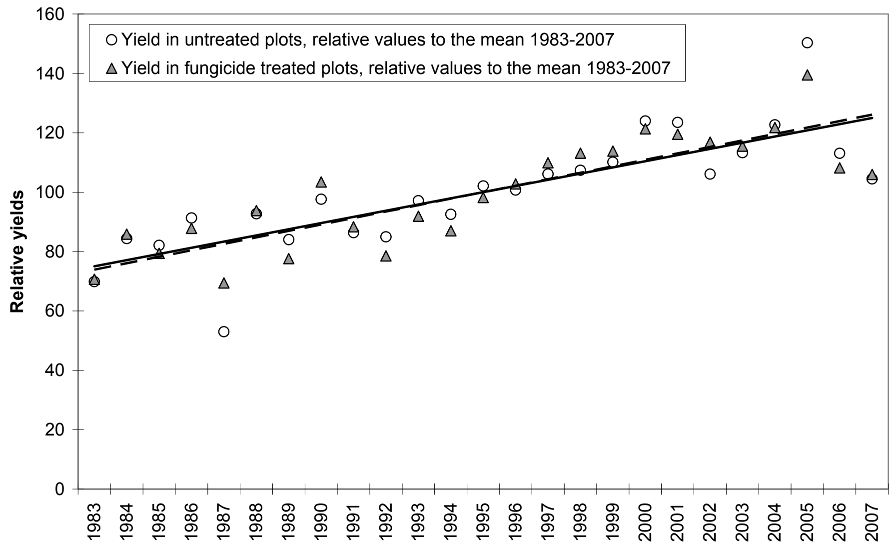

Figure 18. Annual mean yields relative to the mean yield 1983-2007 in untreated plots and fungicide-treated plots in field trials carried out in southern Sweden. Two regression lines are given, almost concordant for untreated and fungicide-treated plots.

5.2 Grain yield quality

While the quantity of grain produced has increased, the important question is whether this grain is of the same quality as in the past, since if the quality is worse than before then it is debatable whether we have made any progress at all.

The following sections present some yield parameters reported in this thesis (Paper I and III) that can give an indication of grain quality. Fungicide treatment affected TGW and HLW positively, and in exceptional years protein content and Hagberg falling number negatively, as also reported elsewhere (Smith & Gooding, 1999; Gooding *et al.*, 2000; Wang *et al.*, 2004; Gooding, 2007). In Figure 12 it can be seen that fungicide treatment resulted in higher hectolitre weight (HLW, g L^{-1}) in about 50% of the 25 years shown, especially years with high disease pressure, *i.e.* years when fungicide treatment resulted in a high yield increase.

Kernel protein content was slightly reduced due to fungicide treatment (Figure 13), although the difference was statistically significant only in 2002, one year out of 18 and a year with severe disease attacks of septoria tritici blotch in almost all field trials (31% attack on leaf 2 compared with the overall mean 1983-2007 of 16%).

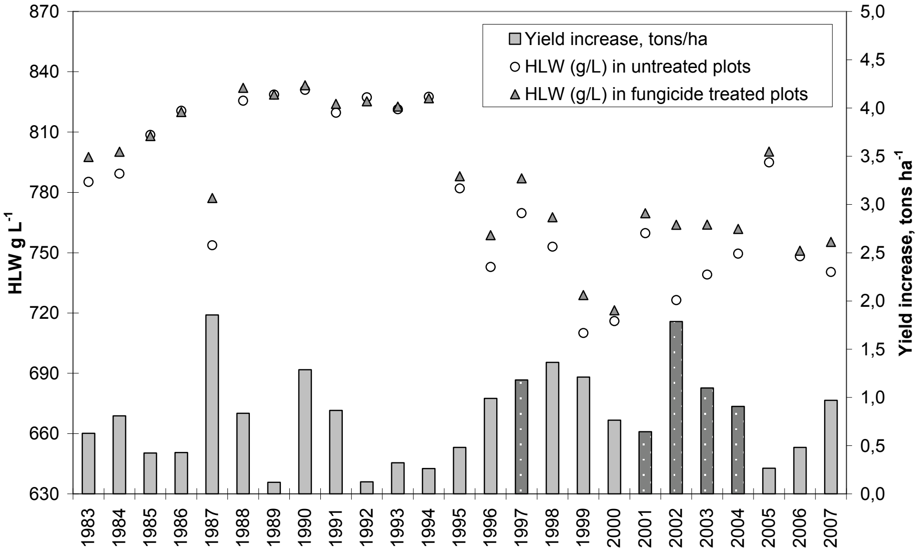

Figure 19. Annual mean hectolitre weight (HLW, g L^{-1}) in untreated and fungicide-treated plots in field trials in southern Sweden 1983-2007 and the resulting grain yield increase. Differences between untreated and fungicide-treated HLW were statistically significant in five years out of 25 (1997, 2001, 2002, 2003 and 2004; darker bars).

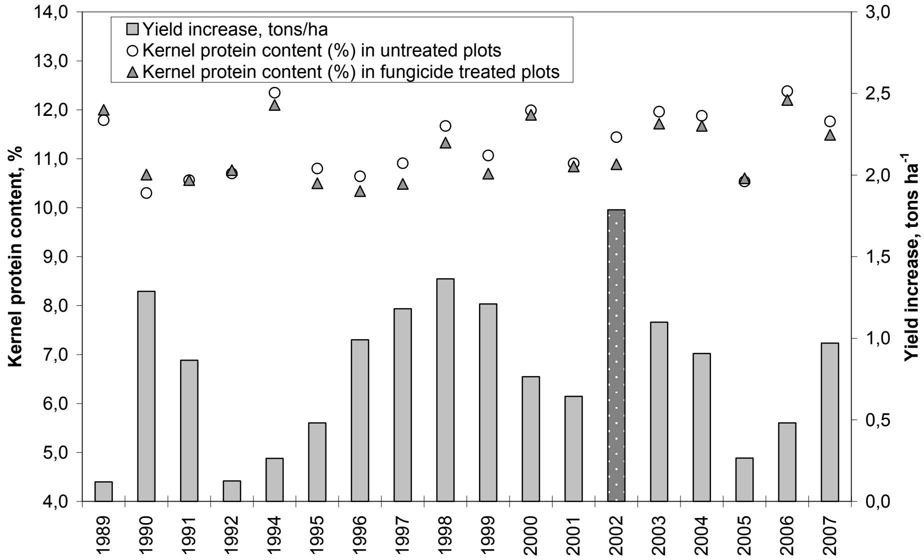

Figure 20. Annual mean kernel protein content (%) in untreated and fungicide-treated plots in field trials in southern Sweden 1989-2007 and the resulting grain yield increase. Differences between untreated and fungicide-treated kernel protein content were statistically significant only in one year (2002; darker bar).

Hagberg falling number was reduced in some years due to fungicide treatment (Figure 14), but this difference was statistically significant only in three years out of 10 (1998, 2001 and 2002).

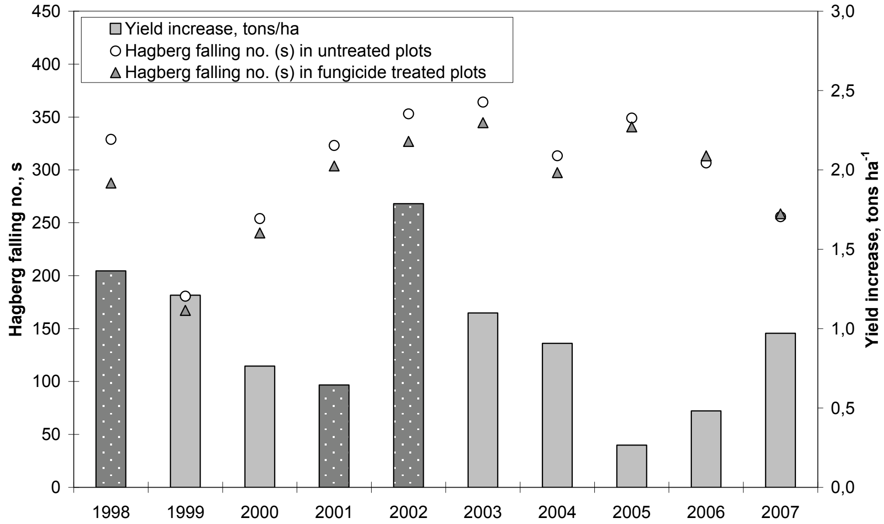

Figure 21. Annual mean Hagberg falling number (s) in untreated and fungicide-treated plots in field trials in southern Sweden 1998-2007 and the resulting grain yield increase. Darker bars indicate years with statistically significant differences.

Reduced kernel protein content and Hagberg falling number due to fungicide treatment can be important in exceptional years but are usually not (Ruske *et al.*, 2004; Gooding, 2007; Wang *et al.*, 2008). In this thesis no marked reduced quality could be seen over years, except for a slight decrease in HLW reported in Paper IV. However, grain quality is much more important than HLW, kernel protein content and Hagberg falling number. If, for example, the kernel protein content of essential minerals has decreased during the selection and plant breeding process, as reported by some scientists (see in the Background section), that is a loss to be considered.

5.3 Plant diseases and their importance

A number of fungal diseases attacked winter wheat in the field trials reported in this thesis (Paper I; Figure 15). An eyespot index was calculated from assessments on samples taken during GS 65-77 as (% weakly attacked tillers)/4 + (% moderately attacked tillers)/2 + (% severely attacked tillers)/1, modified from Scott & Hollins (1974). Eyespot index minus 15 values are given in Figure 15 to get a better representation in relation to the recorded % attack (severity) of the other diseases shown in this schematic figure. Yield is usually affected by eyespot index values of 30 or more and the yield losses are greatest when most lodging occurs. In this thesis annual mean eyespot index exceeded 30 in five years out of 25 (1983, 1990, 1991, 1996 and 2001), but even then infrequently caused lodging. LBDs were common in most years but with a large variation, *e.g.* in 1992-1994 the severity was very low and in 1987 and 2002 very high (Figure 15). Brown rust was more common in the first part of the period and powdery mildew in the latter part, influenced by the proportion of susceptible varieties grown in each period, *e.g.* Kosack was often attacked by brown rust during the late 1980s and beginning of the 1990s. The relationship between disease intensity and proportion susceptible/resistant cultivars is a well-known phenomenon that has been observed in many disease surveys, e.g. King (1977); Polley & Thomas (1991) and Hardwick *et al.* (2001).

Regression analyses revealed that control of LBDs explained 74% of the yield increase achieved by fungicide treatment at GS 45-61, followed by powdery mildew (20%), brown rust (5%) and yellow rust (1%), (Paper I). However, this is a snapshot of the importance of particular diseases for this specific period of time. Climate change, new agricultural practices, *etc.* can dramatically change the order of importance of prevailing diseases and potential diseases. This shift can be fast or slow, such as the rapid change when a rust population adapts to a race-specific resistant cultivar, or the slower change between co-existing leaf blotch diseases caused by *M. graminicola* and *P. nodorum* (Andersson, 1973; Bayles *et al.*, 1990; Bearchell *et al.*, 2005; Fitt *et al.*, 2006; Shaw *et al.*, 2008). Changes during recent decades have been reported, for example, the decline in intensity of stagonospora nodorum blotch and powdery mildew in England and Wales, and the decline in brown rust and increase in powdery mildew in Sweden (Polley & Thomas, 1991; Hardwick *et al.*, 2001; Paper I)

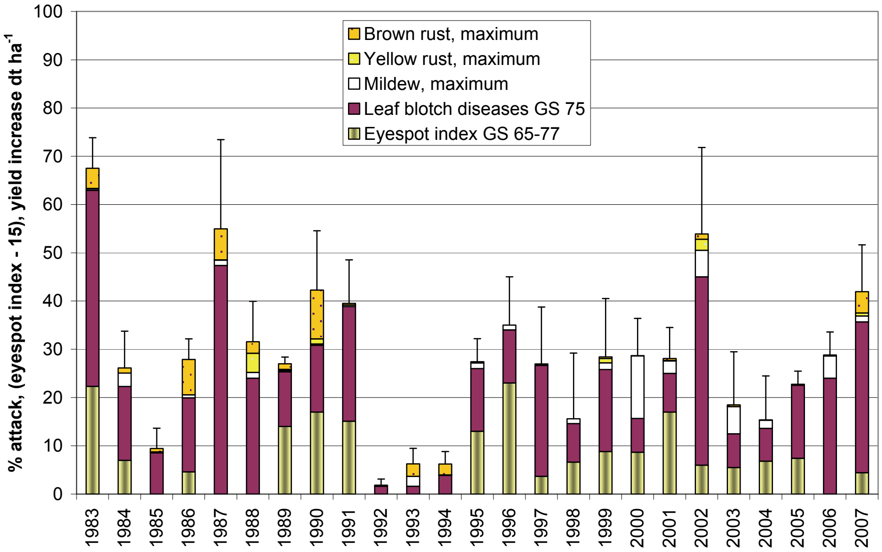

Figure 22. Mean percentage attack of leaf blotch diseases (predominantly septoria tritici blotch and stagonospora nodorum blotch), powdery mildew, yellow rust and brown rust, and eyespot index minus 15. Bars show the yield increase due to a single fungicide treatment at GS 45-61.

5.4 Effect of fungicides

The total yield loss caused by diseases does not correspond to the yield increase achieved by a single fungicide treatment at GS 45-61. Firstly, several diseases are not controlled at all by the fungicides used, and secondly the effect is rarely 100%, especially not with one single fungicide application. Mean fungicide effect during 1983-2007 was 52% (Paper I), which means double yield loss due to fungicide-controllable diseases but there is quite a spread around the regression line, with mean fungicide efficacy <40% in some years and >60% in others (Figure 16). These differences in fungicide efficacy between years can have many explanations, such as prevailing conditions during application, the efficiency of application, fungicide insensitivity and fungicide resistance (Bryson *et al.*, 2006).

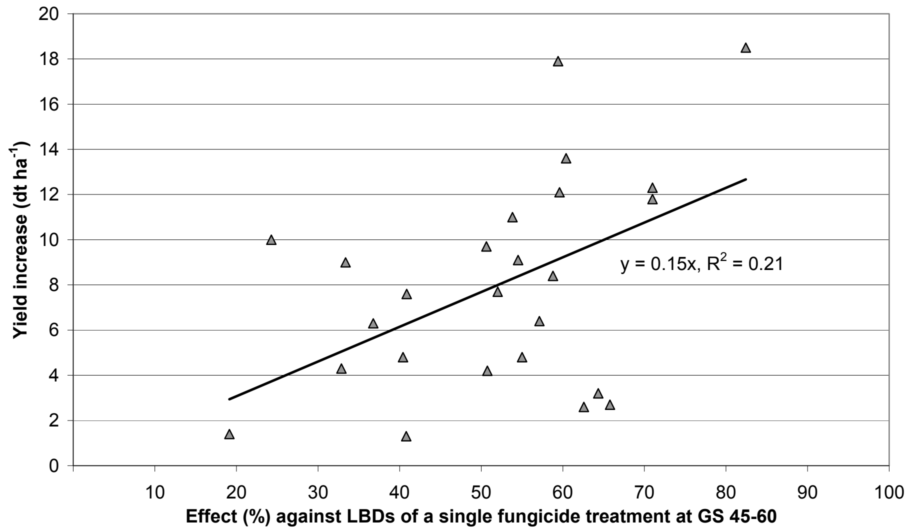

Figure 23. Relationship between yield increases due to a single fungicide treatment at GS 45-61 and the mean annual efficacy against LBDs in field trials carried out in southern Sweden 1983-2007.

5.5 Weather, plant diseases and yield increase

Paper II shows the relationships we found between temperature, precipitation, plant diseases and yield. Our evaluation of monthly precipitation showed May precipitation to be related to leaf blotch diseases and to yield increase due to a single fungicide treatment at GS 45-61 (Figure 17). The importance of spring precipitation has been reported previously in several countries (Shaner & Finney, 1976; Coakley *et al.*, 1985; Emmerman *et al.*, 1988; Murray *et al.*, 1990; Daamen & Stol, 1992; Hansen *et al.*, 1994; Gladders *et al.*, 2001; Pietravalle *et al.*, 2003; Shaw *et al.*, 2008). From Figure 17 it can be seen that the yield increase resulting from control of LBDs is usually small with little precipitation during May. This suggests a negative prognosis, *i.e.* a recommendation of no fungicide application in years when precipitation in May is low.

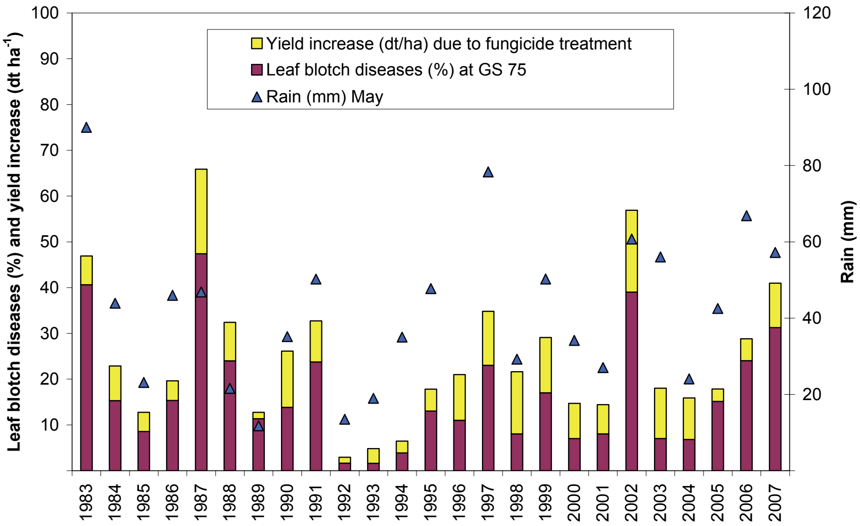

Figure 24. Yield increase due to fungicide treatment, leaf blotch diseases at GS 75 and rain in May and June 1983-2007 in southern Sweden.

It is not only precipitation that is important for the development of plant diseases such as cereal leaf blotch. A number of studies describe the influence of temperature on the *S. tritici* disease cycle (Coakley *et al.*, 1985; Gladders *et al.*, 2001; Pietravalle *et al.*, 2003; Henze *et al.*, 2007). We found mild winters and spring to promote powdery mildew, brown rust and yellow rust. Furthermore, weather factors in the preceding growing season influenced powdery mildew and brown rust (Paper II). Shaw *et al.* (2008) found that previous summer temperature significantly influenced septoria tritici blotch.

In Paper II we showed the potential for using weather data in plant disease prediction, *e.g.* precipitation during tillering, stem elongation and booting for LBDs, while temperature and precipitation in the month prior to sowing were important for powdery mildew and brown rust (Paper II). Our proposed models included three weather factors and R^2 values were in the range 0.41-0.75 and statistically significant. Two LBD models are presented below (*e.g.* MayP is precipitation in May, and JanT is mean temperature in January), one based on disease incidence (I, either attack or no attack) on leaf 1-3 and one based on disease severity (S, % attack) on leaf 3, both at GS 55:

LBDs (I) = 12.4+0.18*MayP-1.2*JanT-0.1*DecP (R^2=0.75, P<0.001)
LBDs (S) = -0.6+0.1*MayP-01*FebP+0.1*AprP (R^2=0.57, P<0.001)

In most of our regression models we found disease incidence assessments to be better than disease severity assessments. The measurements from more than 50 untreated field plots per year in this study are almost certainly more representative of southern Sweden than the severity measurements from a few field trials each year. Relationships between disease incidence and severity have been found in wheat diseases such as powdery mildew, brown rust, LBDs and eyespot (James & Shih, 1973; Seem, 1984; Shaw & Royle, 1987; Fitt *et al.*, 1988; McRoberts *et al.*, 2003; Hughes *et al.*, 2004). In our investigation we found a significant correlation between incidence and severity for LBDs, brown rust and eyespot, but not for yellow rust and powdery mildew as reported in another study (James & Shih, 1973). However, incidence measurements are to be preferred as they are faster to perform, more accurate and less variable, especially if several observers are involved.

5.6 Economics

There are many benefits of using pesticides (Cooper & Dobson, 2007). However, the profitability of using fungicides has been discussed (Cook & King, 1984; Cook & Jenkins, 1988). It is a well-known fact that yield increases due to fungicide treatment are highly variable, following variations in disease severity. Thus, supervised control, decision-making rules and decision support systems are to be preferred, not least to save money when it is not profitable to apply fungicides (Zadoks, 1984; Fabre *et al.*, 2007). In a thorough economic evaluation we showed that a single fungicide treatment at GS 45-61 is somewhat overvalued, especially as seen over the whole 25 year-period studied here (Paper III). The mean net return was negative in 10 years and less than 50% of the entries were profitable to treat in 11 years. During 1995-2007 fungicide use was more profitable, 21 € ha^{-1} compared with 3 € ha^{-1} 1983-1994. An adaptation of the fungicide dose, from 0.8 L ha^{-1} to the estimated optimal 0.66 L ha^{-1}, gained 3 € ha^{-1}, a net return of 24 € ha^{-1} instead of 21 € ha^{-1}.

5.7 Site factors and agricultural practices

Soil factors such as organic matter, clay content, nutrients, soil pH *etc.* as well as agricultural practices such as crop rotation, timing and dose of fertilizers have an impact on yield, plant diseases and interactions among these (Cowling, 1978; Broscious *et al.*, 1985; Murray *et al.*, 1990; Wiese & Veseth, 1991; Rodgers-Gray & Shaw, 2000; Neumann *et al.*, 2004; Walters

& Bingham, 2007). In Paper IV we looked at the relationships among yield and plant diseases versus soil factors and agricultural practices.

Significant changes in many factors were observed over the period 1983-2007, *e.g.* in soil organic matter, soil pH, sand and silt fraction, total nitrogen, day of sowing, Julian day of GS 55, Julian day of spraying, GS at application time, Julian day of harvest and number of days from sowing to harvest. Furthermore, we found significant correlations and interactions among many of these factors. During this time period, cultivars, crop rotation and type of fungicides have changed over the period (Paper I, Paper IV). The soil organic matter decreased by 0.6 percentage units from the first five-year period to the last while soil pH has increased significantly by 0.4 units. A decrease over the total 25-year period was found for Julian day of GS 55. Harvest time was at least one week later in the beginning of the period as compared with the end of the period, and the time from sowing to harvest almost two weeks longer.

In this study, yield was negatively correlated with soil organic matter. In contrast, kernel protein content and powdery mildew were positively correlated with soil organic matter (Paper IV). Furthermore, yield was positively correlated with phosphorus, and powdery mildew negatively with clay content. Other investigations have reported correlations between soil factors and diseases (Van Loon *et al.*, 1998; Wiese *et al.*, 2003), but we found no significant correlations in our data set. In our study, yield, kernel protein content, increase in yield due to fungicide treatment, powdery mildew and LBDs were positively correlated with total nitrogen, which are well-known relationships (Johnston *et al.*, 1979; Howard *et al.*, 1994; Leitch & Jenkins, 1995; Olesen *et al.*, 2003; Walters & Bingham, 2007).

Timing factors affected yield and diseases, such as Julian GS 55 day, *e.g.* leaf blotch diseases at maximum attack were negatively correlated with Julian day of GS 55, and positively with brown rust (Paper IV), almost certainly due to epidemiological and environmental factors (King, 1977; Arama *et al.*, 1999; Gladders *et al.*, 2001; Simón *et al.*, 2005). The incidence of powdery mildew, yellow rust and brown rust changed over the years; powdery mildew increased, yellow rust fluctuated and brown rust decreased. Changes in the proportion of susceptible and resistant cultivars and weather explained differences within and between years (Larsson *et al.*, 2005; Paper I and II).

Crop rotation limits the build-up of pathogen populations, and accurate crop sequencing contributes to maintaining soil fertility (Bockus & Claassen, 1992; Olofsson, 1993; Bailey *et al.*, 2001; Sieling *et al.*, 2007; Fernandez *et al.*, 2009). In comparison with wheat as pre-crop, rape, peas and cereals other than wheat yielded 1.8, 1.5 and 1.3 tons ha^{-1}, respectively, more than two-year wheats in fungicide-treated plots. In untreated plots, the corresponding difference to wheat as pre-crop was 1.6, 1.3 and 1.0 tons ha^{-1}, respectively. Fungicide treatment against foliar diseases was not as beneficial as a favourable pre-crop (Paper IV). In our study the differences in the intensity of plant diseases between different pre-crops were small, which is in agreement with Bailey *et al.* (2001) who found rotation to have a limited impact on wheat disease severity and the prevalence of fungal species relative to the environment.

6 Conclusions and future implementations

The reduction in yield due to plant diseases on winter wheat in southern Sweden can be significant as shown in this thesis. However, the variation in the intensity of different diseases and the yield gain due to fungicide treatment proved to be very large, both in different fields within years and between years. The variation was explained by differences in weather (precipitation and temperature), site factors and agricultural practices. A thorough economic evaluation showed that it is rather often not profitable to apply a fungicide treatment against stem-base and foliar diseases. An important conclusion from this thesis is that more information about factors that influence the build-up of epiphytotics urgently needs to be obtained, through well-planned regional surveys that capture essential data on plant diseases, factors affecting these diseases and assessments of disease-induced crop losses. This information can then be incorporated into effective plant protection strategies that coordinate and optimise control methods. Unfortunately there will always be unknown future events that are impossible to forecast. Risk analysis based on precise and long-term results will make risk-based decision support systems feasible.

Consumers and society are likely to become increasingly important, powerful and demanding. Accordingly, legislation and regulations will support Integrated Pest Management (IPM), plant health management and sustainable agriculture. The dependency on only fungicides or only resistant varieties will probably be reduced, and plant protection will comprise active efforts to increase diversity, improve the crop rotation or avoid poor crop rotations and monocultures, varieties resistant to prevailing diseases, use of appropriate cultural methods and only supervised and prescribed fungicide use (Figure 18, 19).

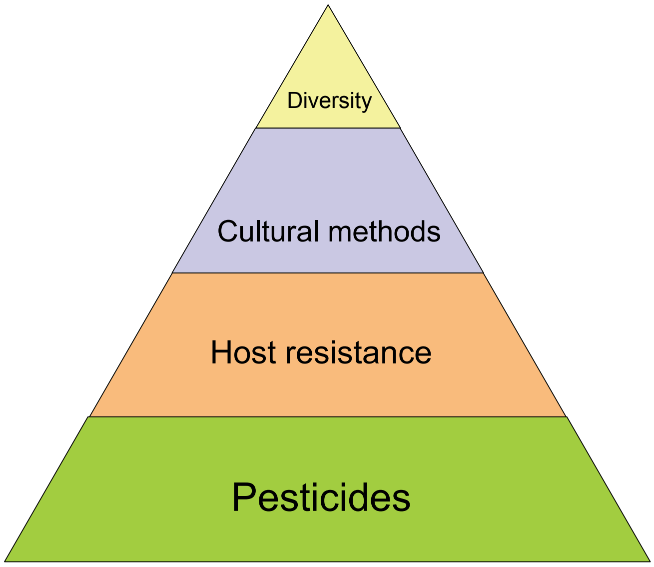

Figure 25. Plant protection today with a high dependency on pesticides.

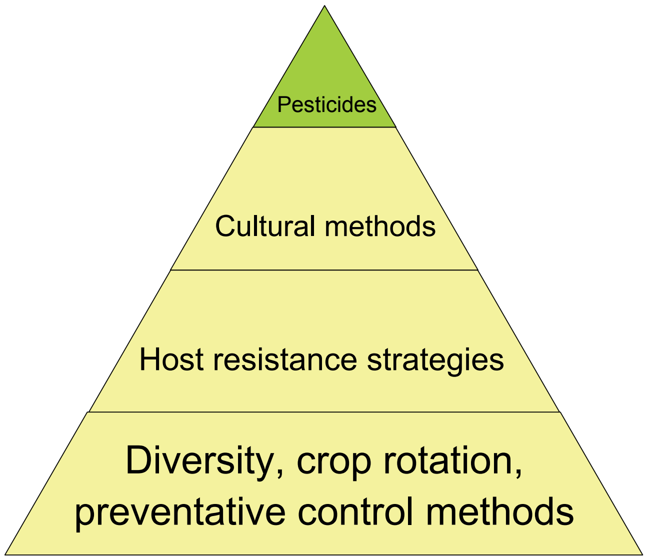

Figure 26. Desirable plant protection with the focus on control methods other than pesticides.

Effective, safe and persistent control strategies can only be devised if control methods are coordinated and co-optimized (Figure 20). IPM ought to be the first and natural choice in plant protection but as pesticides are easily accessible and effective this is not usually the case. The understanding of yield constraints is fundamental in IPM, plant health management and sustainable agriculture and more than one control method may be needed. Increased biodiversity and the right choices of crop rotation, variety and agricultural practices are likely to become important tools in controlling plant diseases if fungicide use is restricted or prohibited in the future. The pieces in the IPM jigsaw puzzle (Figure 20) still have to be assembled, and a lot of research is needed before the desirable development expressed in Figure 19 can be reached. Without large investments on IPM, fungicides will continue to be the corner-stone in plant protection for many years. DNA methodology such as real-time PCR will be increasingly useful in plant protection biology, *e.g.* to identify, quantify and observe changes in fungal populations. The effect of control measures can now be more accurately evaluated. Traditional surveys and field experiments supported by DNA methodology will increase our knowledge of the many interactions taking place in the field. To be applicable IPM needs interdisciplinary action between disciplines such as plant protection biology, plant physiology, meteorology, risk analysis, economics and sociology.

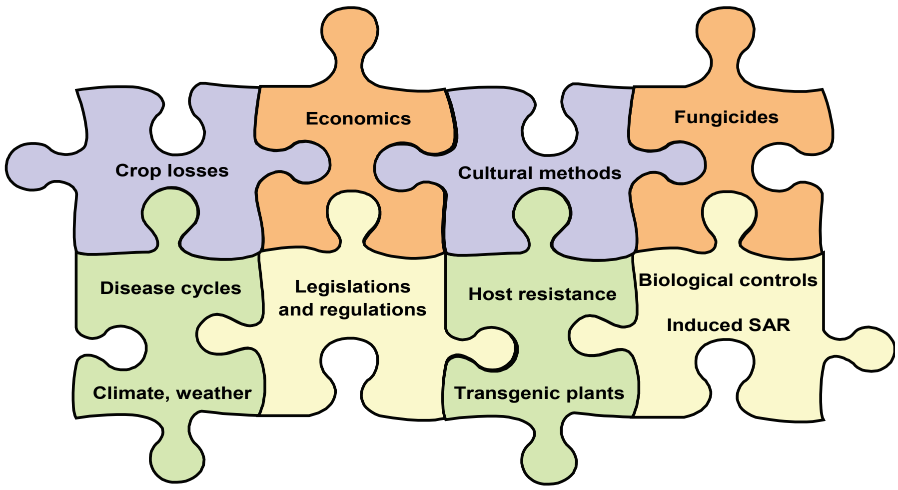

Figure 27. Durable plant protection strategies rely on the integration of many control measures.

6.1 Overall conclusions

6.1.1 Main conclusions

- The results demonstrate the potential and limits of fungicides and the need for supervised control strategies including factors affecting disease, yield and interactions (Paper I).
- The results confirm that weather data can be successfully used in wheat disease prediction models (Paper II).
- Improved decision support systems in a holistic framework based on sound economics are urgently needed (Paper III).
- The role of site factors and agricultural factors is complex but some factors, such as pre-crop and dose of nitrogen, can probably be used in plant disease warning and prediction models (Paper IV).

6.1.2 Detailed conclusions

- Yield and plant diseases in winter wheat are vary widely between years and between fields within years.
- In 1983-2005 by far the most important diseases were the leaf blotch diseases (LBDs including septoria tritici blotch, stagonospora nodorum blotch and tan spot), and of these septoria tritici blotch was the most important.
- In the period 1983-2005, yield increased from 6 tons ha^{-1} to 12 tons ha^{-1} in field trials.
- In the period 1977-2002, single eyespot treatment improved yield by approximately 320 kg ha^{-1}, mainly due to occasional years with severe attacks.
- Single treatment at GS 45-61 against foliar diseases improved yield by 660 kg ha^{-1} in the period 1983-1994 and by 970 kg ha^{-1} in the period 1995-2005.
- In the period 1983-2005, an additional treatment at GS 30-40 against foliar diseases improved yield approximately by 250 kg ha^{-1}.
- Weather factors, the driving forces in plant disease development, vary widely between years and between fields within years.
- The weather variables air temperature and precipitation (rain) explained more than 50% of the variation between years regarding yield increase due to fungicide treatment, thousand grain weight, hectolitre weight, LBDs, brown rust, yellow rust and eyespot.
- The weather variables air temperature and precipitation (rain) explained less than 50% of the variation between years regarding yield level and powdery mildew.

- Precipitation in May was the factor most consistently related to LBD disease intensity.
- Weather factors in the preceding growing season influenced growth stage, powdery mildew and brown rust.
- Mild winters and springs favoured the biotrophs, *i.e.* powdery mildew, brown rust and yellow rust.
- Statistically significant correlations between incidence and severity were found for LBDs, brown rust and eyespot, but not for yellow rust and powdery mildew.
- Regression models with disease incidence as the dependent variable generally had higher R^2-values and lower P-value than models with disease severity as the dependent variable.
- The mean net return from fungicide use was no more than 12 € ha^{-1} over the 25 years (2008 grain prices and costs used in calculations).
- The mean net return was negative in 10 years, and in 11 years, less than 50% of the entries were profitable to treat.
- Fungicide use was in fact more profitable (mean net return 21 compared with 3 € ha^{-1}) during the latter part of the study period (1995-2007) than in the earlier part (1983-1994).
- Wheat as pre-crop to wheat gave 1.8 and 1.6 tons ha^{-1} lower yield than rape as pre-crop in untreated and fungicide-treated plots, respectively. Fungicide treatment against foliar diseases was not as beneficial as a favourable pre-crop.
- The intensity of leaf blotch diseases, powdery mildew and yellow rust increased with higher total nitrogen levels.
- The intensity of leaf blotch diseases was smaller in years when GS 55 was reached later rather than earlier.
- The intensity of powdery mildew increased with increasing organic matter content and decreased with increasing clay content.
- The intensity of brown rust was higher when GS 55 was reached later rather than earlier.
- Overall, the differences in intensity of foliar plant diseases between different pre-crops were small.

6.2 Recommendations

Control methods against plant diseases, such as host plant resistance, cultural methods and fungicides, can be better be exploited in integrated pest management strategies. To become reality, interdisciplinary research must be carried out to synchronise available control measures.

References

Agrios, G.N. 2005. *Plant pathology*. Fifth edition. Elsevier Academic Press.

Agriwise (2009). www.agriwise.org (accessed March 2009).

Åkerberg, E. 1986. Svalöf 1886-1986. A historical survey of the breeding research. In *Svalöf 1886-1986. Research and Results in Plant Breeding*, 9-34.

Almquist, C., Lerenius, C., Filipsson, C., Jonsson, A. 2008. Bestämning av förekomst av patogena svampar i vete med PCR-teknik. SLU, Avdelningen för precisionsodling, *Rapport* 18, 44 s. http://po-mv.slu.se.

Andersson, K. 1973. Gulrost-angreppen i Skåne 1972. *Växtskyddsnotiser* 37, 27-32.

Andersson, K., Larsson, H., Nilsson, I., Wiik, L. 1986. Försök med odlingssystem i höstvete i Skåne 1978-1985 (Engl. summary). Ogräs- och växtskyddskonferenserna 1986. Gemensam och växtskyddsdel. *Växtskyddsrapporter. Jordbruk* 39, 211-233.

Arama, P.F., Parlevliet, J.E., van Silfhout, C.H. 1999. Heading date and resistance to septoria tritici blotch in wheat not genetically associated. *Euphytica* 106, 63-68.

Arraiano, L.S., Brown, J.K.M. 2006. Identification of isolate-specific and partial resistance to septoria tritici blotch in 238 European wheat cultivars and breeding lines. *Plant Pathology* 55, 726-738.

Arraiano, L.S., Balaam, N., Fenwick, P.M., Chapman, C., Feuerhelm, D., Howell, P., Smith, S.J., Widdowson, J.P., Brown, J.K.M. 2009. Contributions of disease resistance and escape to the control of septoria tritici blotch of wheat. *Plant Pathology* 58, 910-922.

Audsley, E., Milne, A., Paveley, N. 2005. A foliar disease model for use in wheat disease management decision support systems. *Annals of Applied Biology* 147, 161-172.

Aust, H.-J., Hoyningen-Huente v., J. 1986. Microclimate in relation to epidemics of powdery mildew. *Annual Review of Phytopathology* 24, 491-510.

Austin, R.B. 1999. Yield of winter wheat in the United Kingdom: Recent advances and prospects. *Crop Science* 39, 1604-1610.

Babij, J., Zhu, Q., Brain, P., Hollomon, D.W. 2000. Resistance risk assessment of cereal eyespot, *Tapesia yallundae* and *Tapesia acuformis*, to the anilinopyrimidine fungicide, cyprodinil. *European Journal of Plant Pathology* 106, 895-905.

Bagge, M., Hovmøller, M.S., Andersen, S.B. 2008. Efficacy of gene pyramiding of identified powdery mildew- and yellow rust- resistance genes in elite breeding material of hexaploid wheat. In Marker assisted selection for disease resistance in wheat, 85-105. PhD *Thesis* Merethe Bagge *Copenhagen University Denmark.*

Bahat, A., Gelernter, I., Brown, M.B., Eyal, Z. 1980. Factors affecting the vertical progression of Septoria leaf blotch in short-statured wheats. *Phytopathology* 70, 179-184.

Bailey, K.L., Gossen, B.D., Lafond, G.P., Watson, P.R., Derksen, D.A. 2001. Effect of tillage and crop rotation on root and foliar diseases of wheat and pea in Saskatchewan from 1991 to 1998: Univariate and multivariate analyses. *Canadian Journal of Plant Science* 81, 789-803.

Bakker, M.M., Govers, G., Ewert, F., Rounsevell, M., Jones, R. 2005. Variability in regional wheat yields as a function of climate, soil and economic variables: Assessing the risk of confounding. *Agriculture, Ecosystems and Environment* 110, 195-209.

Baltazar, B.M., Scharen, A.L., Kronstad, W.E. 1990. Association between dwarfing genes ´Rht1´ and ´Rht2´and resistance to Septoria tritici Blotch in winter wheat (Triticum aestivum L. em Thell). *Theoretical and Applied Genetics* 79, 422-426.

Bancal, M-O., Robert, C., Ney, B. 2007. Modelling wheat growth and yield losses from late epidemics of foliar diseases using loss of green leaf area per layer and pre-anthesis reserves. *Annals of Botany* 100, 777-789.

Bariana, H.S., Brown, G.N., Bansal, U.K., Miah, H., Standen, G.E., Lu, M. 2007. Breeding triple rust resistant wheat cultivars for Australia usinng conventional and marker-assisted selection technologies. *Australian Journal of Agricultural Research* 58, 576-587.

Bartlett, D.W., Clough, J.M., Godwin, J.R., Hall, A.A., Hamer, M., Parr-Dobrzanska, B. 2002. The strobilurin fungicides. *Pest Management Science* 58, 649-662

Bateman; G.L. 2002. Long-term performance of fungicides applied to control eyespot in winter wheat. *Annals of applied Biology* 141, 29-33.

Bateman, G.L., Edwards, S.G., Marshall, J., Morgan, L.W., Nicholson, P., et al. 2000. Effects of cultivar and fungicides on stem-base pathogens, determined by quantitative PCR, and on diseases and yield of wheat. *Annals of applied Biology* 137, 213-221.

Bayles, R.A., Parry, D.W., Priestley, R.H. 1985. Resistance of winter wheat varieties to *Septoria tritici*. *J. natn. Inst. agric. Bot.* 17, 21-26.

Bayles, R.A., Channel, M.H., Stigwood, P.L. 1990. Yellow rust of wheat. *U.K. Cereal Pathogen Survey. 1989 Annual Report*, 11-17.

Bayles, R.A. 1991. Research note: Varietal resistance as a factor contributing to the increased importance of *Septoria tritici* Rob. And Desm. In the UK wheat crop. *Plant Varieties and Seeds* 4, 177-183.

Bayles, R.A. 1997. Disease development – the interaction of variety resistance and pathogen. *Aspects of Applied Biology* 50, 249-254.

Bayles, R. 1999. The interaction of strobilurin fungicides with cereal varieties. *Plant Varieties and Seeds* 12 (2), 129-140.

Bayles, R.A., Flath, K., Hovmøller, M.S., de Vallavieille-Pope, C. 2000. Breakdown of the *Yr17* resistance to yellow rust of wheat in northern Europe. *Agronomie* 20, 805-811.

Bayles, R.A., Stigwood, P.L. 2001. Yellow rust of wheat. *U.K. Cereal Pathogen Survey. 2000 Annual Report*, 28-36.

Bearchell, S.J., Fraaije, B.A., Shaw, M.W., Fitt, B.D.L. 2005. Wheat archive links long-term fungal pathogen population dynamics to air pollution. *Proceedings of the National Academy of Sciences of the United States of America* 102, 5438-5442.

Bélanger, R.R., Benhamou, N., Menzies, J.G. 2003. Cytological evidence of an active role of silicon in wheat resistance to powdery mildew (*Blumeria graminis* f. sp. *tritici*). *Phytopathology* 93, 402-412.

Benizri, E., Projetti, F. 1992. Mise au point d'un modèle de simulation de la rouille brune du blé. *Agronomie* 12, 97-104.

Bingham, I.J., Walters, D.R., Foulkes, M.J., Paveley, N.D. 2009. Crop traits and the tolerance of wheat and barley to foliar diseases. *Annals of Applied Biology* 154, 159-173.

Bockus, W.W., Claassen, M.M. 1992. Effects of crop rotation and residue management practices on severity of tan spot of winter wheat. *Plant Disease* 76, 633-636.

Bockus, W.W., Shroyer, J.P. 1998. The impact of reduced tillage on soilborne plant pathogens. *Annual Review of Phytopathology* 36, 485-500.

Bockus, W.W., Appel, J.A., Bowden, R.L., Fritz, A.K., Gill, B.S., Martin, T.J., Sears, R., Seifers, D.L., Brown-Guedira, G.L., Eversmeyer, M.G. 2001. Success stories: Breeding for wheat disease resistance in Kansas. *Plant Disease* 85, 453-461.

Borlaug, N.E. 1981. Increasing and stabilizing food production. In *Plant Breeding II*, 467-492 (Frey, K.J. ed.). CIMMYT. ISBN 0-8138-1550-9.

Borlaug, N. 2007. Feeding a hungry world. *Science* 318, 359.

Boshoff, W.H.P., Pretorius, Z.A., van Niekerk, B.D. 2002. Establishment, distribution, and pathogenicity of *Puccinia striiformis* f. sp. *tritici* in South Africa. *Plant Disease* 86, 485-492.

Bourke, A., Lamb, H. 1993. The spread of potato blight in Europe in 1845-6 and the accompanying wind and weather patterns. Published by *the Meteorological Service Glasnevin Hill, Dublin* 9, pp. 66.

Brancourt-Hulmel M, Doussinault G, Lecomte C, Bérard P, Le Buanec B, Trottet M. 2003. Genetic improvement of agronomic traits of winter wheat cultivars released in France from 1946 to 1992. *Crop Science* 43, 37-45.

Bray, E.A., Bailey-Serres, J. Weretilnyk, E. 2000. Responses to abiotic stresses. In *Biochemistry & Molecular biology of plants* (eds. Buchanan, B.B., Gruissem, W, Jones, R.L.), 1158-1203. American Society of Plant Physiologists.

Broscious, S.C., Frank, J.A., Frederick, J.R. 1985. Influence of winter wheat management practices on the severity of powdery mildew and septoria blotch in Pennsylvania. *Phytopathology* 75, 538-542.

Brown, J.K.M., Hovmøller, M.S. 2002. Aerial dispersal of pathogens on the global and continental scales and its impact on plant disease. *Science* 297, 537-541.

Browning, J.A. 1998. One phytopathologist's growth through IPM to holistic plant health. The key to approaching genetic yield poptential. *Annual Review of Phytopathology* 36, 1-24.

Browning, J.A. and Frey, K.J. 1969. Multiline cultivars as a means of disease control. *Annual Review of Phytopathology* 7, 355-382.

Brunner, P.C., Stefanato, F.L., McDonald, B.A. 2008. Evolution of the *CYP51* gene in *Mycosphaerella graminicola*: evidence for intragenic recombination and selective replacement. *Molecular Plant Pathology* 9, 305-316.

Bryson RJ, Paveley ND, Clark WS, Sylvester-Bradley R, Scott RK. 1997. Use of in-field measurements of green leaf area and incident radiation to estimate the effects of yellow rust epidemics on the yield of winter wheat. *European Journal of Agronomy* 7, 53-62.

Bryson, R.J., Burnett, F.J., Foster, V., Fraaije, B.A., Kennedy, R. (eds.) 2006. Fungicide resistance: Are we winning the battle but loosing the war. *Aspects of Aspplied Biology* 78.

Buchenau, G.W. 1975. Relationship between yield loss and area under the wheat stem rust and leaf progress curves. *Phytopathology* 65, 1317-1318.

Bürger, J., de Mol, F., Gerowitt, B. 2008. The "necessary extent" of pesticide use - Thoughts about a key term in German pesticide policy. *Crop Protection* 27, 343-351.

Burke, J.J., Dunne, B. 2008. Field testing of six decision support systems for scheduling fungicide applications to control *Mycosphaerella graminicola* on winter wheat crops in Ireland. *Journal of Agricultural Science* 146, 415-428.

Burnett, F.J. 2005. Validation of a risk assessment method to identify wheat crops at risk from eyespot. *HGCA* (The Home-Grown Cereals Authority, UK) *project report* no. 347, part 2, 27 pp.

Burnett, F.J., Hughes, G. 2004. The development of a risk assessment method to identify wheat crops at risk from eyespot. *HGCA* (The Home-Grown Cereals Authority, UK) *project report* no. 347, 67 pp.

Cadle, M.M., Murray, T.D. 1997. Identification of resistance to *Pseudocercosporella herpotrichoides* in *Triticum monococcum*. *Plant Disease* 81, 1181-1186.

Campbell, C.L., Madden, L.V. 1990. *Introduction to plant disease epidemiology*.

Cavelier, A., Cavelier, N., Colas, A.Y., Montfort, F., Lucas, P. 1998. ITITECH: a survey to improve the evaluation of relationships between cultural practices and cereal disease incidence. *The 1998 Brighton Conference – Pest & Diseases* vol. 3, 1023-1028.

Chapman, N.H., Burt, C., Dong, H., Nicholson, P. 2008. The development of PCR-based markers for the selection of eyespot resistance genes *Pch1* and *Pch2*. *Theoretical and Applied Genetics* 117, 425-433.

Chartrain, L., Brading, P.A., Makepeace, J.C., Brown, J.K.M. 2004. Sources of resistance to septoria tritici blotch and implications for wheat breeding. *Plant Pathology* 53, 454-460.

Chen, X.M. 2005. Epidemiology and control of stripe rust (*Puccinia striiformis* f. sp. *tritici*) on wheat. *Canadian Journal of Plant Pathology* 27, 314-337.

Chen, X.M. 2007. Challenges and solutions for stripe rust control in the United States. *Australian Journal of Agricultural Research* 58, 648-655.

Chen, X., Line, R.F., Leung, H. 1995. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f.sp. *hordei* to other rusts. *Phytopathology* 85, 1335-1342.

Chester, K.S. 1950. Plant disease losses: Their appraisal and interpretation. *The Plant Disease Reporter*, supplement 193, pp. 362.

Chiarappa, L., Chiang, H.C., Smith, R.F. 1972. Plants pests and diseases: Assessment of crop losses. *Science* 176, No. 4036, 769-773.

Chiarappa, L. (ed.) 1981. *Crop loss assessment methods - Supplement 3, Commonwealth Agricultural Bureaux FAO*, 123 pp.

Clarkson, J.D.S. 1981. Relationship between eyespot severity and yield loss in winter wheat. *Plant Pathology* 30, 125-131.

Clarkson, J.D.S., Cook, R.J. 1983. Effect of sharp eyespot (*Rhizoctonia cerealis*) on yield loss in winter wheat. *Plant Pathology* 32, 421-428.

Clifford, B.C., Harris, R.G.. 1981. Controlled environment studies of the epidemic potential of *Puccinia recondita* f.sp. *tritici* on wheat in Britain. *Transactions of the British mycological Society* 77, 351-358

Coakley, S.M., McDaniel, L.R., Shaner, G. 1985. Model for predicting severity of Septoria tritici blotch on winter wheat. *Phytopathology*, 75, 11, 1245-1251.

Coakley, S.M. 1988. Historical weather data: Its use in epidemiology. In *Plant Disease Epidemiology*, ed. K Leonard and W Fry, Vol. II, 54-83. New York: MacMillan Publishing Co.

Coakley, S.M., Line, R.F., McDaniel, L.R. 1988. Predicting stripe rust severity on winter wheat using an improved method for analysing meteorological and rust data. *Phytopathology* 78, 543-550.

Colbach, N., Saur, L. 1998.Influence of crop management on eyespot development and infection cycles of winter wheat. *European Journal of Plant Pathology* 104, 37-48.

Cook, J.W., Kettlewell, P.S., Parry, D.W. 1993. Control of *Erysiphe graminis* and *Septoria tritici* with foliar applied potassium chloride. *Journal of the Science of Food and Agriculture* 63, 126.

Cook, R.J. 1977. Effect of timed fungicide sprays on yield of winter wheat in relation to Septoria infection periods. *Plant Pathology* 26, 30-24.

Cook, R.J. 1980. Effects of late-season fungicide sprays on yield of winter wheat. *Plant Pathology* 29, 21-27.

Cook, R.J., King, J.E. 1984. Loss caused by cereal diseases and the economics of fungicidal control. In *Plant Diseases: infection, damage and loss*, 237-245. Blackwell Scientifique Publications, Oxford.

Cook, R.J. 1987. Effects of late season fungicides on winter wheat quality. Aspects of Applied Biology 15. *Cereal quality*, 417-423.

Cook, R.J., Jenkins J.E.E. 1988. The contribution and value of pesticides to disease control in cereeals. In *Control of plant diseases: costs and benefits* (eds. Clifford BC, Lester E), 3-12.

Cook, R.J., Thomas, M.R. 1990. Influence of site factors in responses of winter wheat to fungicide programmes in England and Wales, 1979-1987. *Plant Pathology* 39, 548-557.

Cook, R.J., Polley, R.W., Thomas, M.R. 1991. Disease-induced losses in winter wheat in England and Wales 1985-1989. *Crop Protection*, 504-508.

Cook, R.J., Hims, M.J., Vaughan, T.B. 1999. Effects of fungicide spray timing on winter wheat disease control. *Plant Pathology* 48, 33-50.

Cooke, B.M. 1998. Disease assessment and yield loss. In *The epidemiology of plant disease*, ed. Jones G, 42-72.

Cooper, J., Dobson, H. 2007. The benefits of pesticides to mankind and the environment. *Crop Protection* 26, 1337-1348.

Cowling, E.B. 1978. Agricultural and forest practices that favour epidemics. In *Plant Disease. An Advanced Treatise* vol. II, How Diseases develops in populations, 361-381.

Crous, P.W., Groenewald, J.Z., Gams, W. 2003. Eyespot of cereals revisted: ITS phylogeny reveals new species relationships. *European Journal of Plant Pathology* 109, 841-850.

Cu, R.M., Line, R.F. 1994. An expert advisory system for wheat disease management. *Plant Disease* 78, 209-215.

Cunfer, B.M., Touchton, J.T., Johnson, J.W. 1980. Effects of phosphorus and potassium fertilization on Septoria glume blotch of wheat. *Phytopathology* 70, 1196-1199.

Cunfer, B.M., Ueng, P.P. 1999. Taxonomy and identification of *Septoria* and *Stagonospora* species on small-grain cereals. Annual Review of Phytopathology 37, 267-284.

Cunfer, B.M., Buntin, G.D., Phillips, D.V. 2006. Effect of crop rotation on take-all of wheat in double-cropping systems. *Plant Disease* 90, 1161-1166.

Daamen, R.A. 1989. Assessment of the profile of powdery mildew and its damage function at low disease intensities in field experiments with winter wheat. *Netherlands Journal of Plant Pathology* 95, 85-105.

Daamen, R.A., Stol, W. 1992. Surveys of cereal diseases and pests in the Netherlands. 5. Occurrence of *Septoria* spp. in winter wheat. *Netherlands Journal of Plant Pathology* 98, 369-376.

Daamen, R.A., Stubbs, R.W., Stol, W. 1992. Surveys of cereal diseases and pests in the Netherlands. 4. Occurrence of powdery mildew and rusts in winter wheat. *Netherlands Journal of Plant Pathology* 98, 301-312.

Davidse, L.C., Waard de, M.A. 1984. Systemic fungicides. *Advances in Plant Pathology* vol. 2, 191-257.

Dekker, J., Georgopoulos, S.G. 1982. *Fungicide resistance in crop protection*. Wageningen, the Netherlands.

Delin, S., Nyberg, A., Lindén, B., Ferm, M., Torstensson, G., Lerenius, C., Gruvaeus, I. 2008. Impact of crop protection on nitrogen utilisation and losses in winter wheat production. *European Journal of Agronomy* 28, 361-370.

Delp, C.J. 1986. Pesticide resistance management is a key to effective pest control. *BioScience* 36, 101-102.

DESSAC. 2000. Decision support system for arable crops. LINK, BBSRC and *HGCA Projects*. http://www.dessac.rothamsted.ac.uk/ 2008-06-16.

de Vallavieille-Pope, C., Giosue, S., Munk, L., Newton, A.C., Niks, R.E., Østergård, H., Pons-Kühnemann, J., Rossi, V., Sache, I. 2000. Assessment of epidemiological parameters and their use in epidemiological and forecasting models of cereal airborne diseases. *Agronomie* 20, 715-727.

De Wolf, E.D., Isard, S.A. 2007. Disease cycle approach to plant disease prediction. *Annual Review of Phytopathology* 45, 203-220.

Doussinault, G., Delibes, A., Sanchez-Monge, R., Garcia-Olmedo, F. 1983. Transfer of a dominant gene for resistance to eyespot disease from a wild grass to hexaploid wheat. *Nature* 303, 698-700.

Ellis, J.G., Mago, R., Kota, R., Dodds, P.N. et al. 2007. Wheat rust research at CSIRO. *Australian Journal of Agricultural Research* 58, 507-511.

Emmerman, A., Gustafsson, G., Hedene, K-A., Sigvald, R., Wiik, L. 1988. Prediction of leaf and glume blotch diseases in winter wheat and spring barley. *Växtskyddsnotiser* 52, 112-116.

Ennaifar, S., Makowski, D., Meynard, J.M., Lucas, P. 2007. Evaluation of models to predict take-all incidence in winter wheat as a function of cropping practices, soil, and climate. *European Journal of Plant Pathology* 118, 127-143.

Eriksen, L., Munk, L. 2003. The occurrence of *Mycosphaerella graminicola* and its anamorph *Septoria tritici* in winter wheat during the growing season. *European Journal of Plant Pathology* 109, 253-259.

Eriksen, L., Borum, F., Jahoor, A. 2003. Inheritance and localisation of resistance to *Mycosphaerella graminicola* causing septoria tritici blotch and plant height in the wheat (*Triticum aestivum* L.) genome with DNA markers. *Theoretical Applied Genetics* 107, 515-527.

Eversmeyer, M.G., Kramer, C.L. 1998. Models of early spring survival of wheat leaf rust in the central Great Plains. *Plant Disease* 82, 987-991.

Eversmeyer, M.G., Kramer, C.L. 2000. Epidemiology of wheat leaf and stem in the central Great Plains of the USA. *Annual Review of Phytopathology* 38, 491-513.

Ewaldz, N.A.T. 2000. Radiometric readings as a tool for predicting optimal fungicide dose in winter wheat. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 107, 6, 594-604.

Ewaldz, T., Berg, G. 2008. Svampförsök i höstvete och vårkorn 2007. Skåneförsök 2007. *Försöksringarna och hushållningssällskapen i Skåne, Meddelande* nr 74, 166-174.

Ewaldz, T., Berg, G. 2009. Fungicidförsök i höstsäd 2008. Skåneförsök 2007. *Försöksringarna och hushållningssällskapen i Skåne, Meddelande* nr 75, 128-134.

Eyal, Z., Scharen, A.L., Huffman, M.D., Prescott, J.M. 1985. Global insights into virulence frequencise of *Mycosphaerella graminicola. Phytopathology* 75, 1456-1462.

Eyal, Z. 1999a. Breeding for resistance to Septoria and Stagonospora diseases of wheat. In *Septoria on cereals. A study of pathosystems*, eds. Lucas JA, Bowyer P, Anderson HM, 332-344. CABI Publishing ISBN 0 85199 269 2.

Eyal, Z. 1999b. The septoria tritici and stagonospora nodorum blotch diseases of wheat. *European Journal of Plant Pathology* 105, 629-641.

Fabre, F., Plantegenest, M., Yuen, J. 2007. Finacial benefit of using crop protection decision rules over systematic spraying strategies. *Phytopathology* 97, 1484-1490.

Fajersson, F. 1961. Nitrogen fertilization and wheat quality. *Agri Hortique Genetica* XIX, 1-195.

Fan, M-S., Zhao, F-J., Fairweather-Tait, S.J., Poulton, P.R., Dunham, S.J., McGrath, S.P. 2008. Evidence of decreasing mineral density in wheat grain over the last 160 years. *Journal of Trace Elements in Medicine and Biology* 22, 315-324.

Fernandez, M.R., Pearse, P.G., Holzgang, G., Basnyat, P., Zentner, R.P. 2009. Impacts of agronomic practices on the leaf spotting complex of common wheat in eastern Saskatchewan. *Canadian Journal of Plant Science* 89, 717-730.

Finckh, M.R., Wolfe, M.S. 1998. Diversification strategies. In *The Epidemiology of Plant Diseases*, Jone, D.G. (ed.), 231-259

Finckh, M.R., Gacek, E.S., Goyeau, H., Lannou, C., Merz, U. et al. 2000. Cereal variety and species mixtures in practice, with emphasis on disease resistance. *Agronomie* 20, 813-837.

Fitt, B.D.L., Goulds, A., Polley, R.W. 1988. Eyespot (*Pseudocercosporella herpotrichoides*) epidemiology in relation to prediction of disease severity and yield loss in winter wheat – a review. *Plant Pathology* 37, 311-328.

Fitt, B.D.L., McCartney, H.A., Walklate, P.J. 1989. The role of rain in dispersal of pathogen inoculum. *Annual Review of Phytopathology* 27, 241-270.

Fitt, B.D.L., Huang, Y.-J., Bosch van den, F., West, J.S. 2006. Co existence of related pathogen species on arable crops in space and time. *Annual Review of Phytopathology* 44, 163-182.

Flor, H.H. 1971. Current status of the gene-for-gene concept. *Annual Review of Phytopathology* 9, 275-296.

Folkesson, Ö., 1992. Körskador vid körning i växande gröda. *Faktablad om växtskydd, Jordbruk* 65 J, 4 p. SLU Uppsala.

FRAC 2009. Fungicide Resistance Action Committee. www.frac.info accessed August 2009.

Fraaije, B.A., Lucas, J.A., Clark, W.S., Burnett, F.J. 2003. QoI resistance development in populations of cereal pathogens in the UK. *The BCPC International Congress – Crop Science & Technology*, 689-694.

Fraaije, B.A., Cools, H.J., Fountaine, J., Lovell, D.J., Motteram, J., West, J.S., Lucas, J.A. 2005. Role of ascospores in further spread of QoI-resistant cytochrome b alleles (G143A) in field populations of *Mycosphaerella graminicola*. *Phytopathology* 95, 933-941.

Friedrich, S. 1995. Zur berechnung der konidienverbreitung von *Erysiphe graminis* in natürlich befallenen beständen anhand stündlicher meteorologischer parameter. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 102, 337-347

Fry,W.E. 1978. Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. *Phytopathology* 68, 1650-1655.

García-Guzmán, G., Morales, E. 2007. Life-history strategies of plant pathogens: Distribution patterns and phylogenetic analysis. *Ecology* 88, 589-596.

Garvin, D.F., Welch, R.M., Finley, J.W. 2006. Historical shifts in the seed mineral micronutrient concentration of US hard red winter wheat germplasm. *Journal of the Science of Food and Agriculture* 86, 2213-2220.

Geagea, L., Huber, L, Sache, I. 1999. Dry-dispersal and rain-splash of brown (*Puccinia recondita* f.sp. *tritici*) and yellow (*P. striiformis*) rust spores from infected wheat leaves exposed to simulated raindrops. *Plant Pathology* 48, 472-482.

Gianessi, L., Reigner, N. 2006. The importance of fungicides in U.S. crop production. *Outlooks on Pest Management*, 209-213.

Gjaerum H.B., Leijerstam, B., Ólafsson, S., Skou, J.P., Ylimäki, A. 1985. *Nordic names of plant diseases and pathogens*. NJF. Det kgl. Danske Landhusholdningsselskap.

Gladders, P., Paveley, N.D., Barrie, I.A., Hardwick, N.V., Hims, M.J., Langton, S., Taylor, M.C. 2001. Agronomic and meteorological factors affecting the severity of leaf blotch caused by *Mycosphaerella graminicola* in commercial wheat crops in England. *Annals of applied Biology* 138, 301-311.

Gladders, P., Langton, S., Barrie, I.A., Hardwick, N.V., Taylor, M.C., Paveley, N.D. 2007. The importance of weather and agronomic factors for the overwinter survival of yellow rust (*Puccinia striiformis*) and subsequent disease risk in commercial wheat crops in England. *Annals of applied Biology* 150, 371-382.

Glawe, D.A. 2008. The powdery mildews: A review of the world's most familiar (yet poorly known) plant pathogens. *Annual Review of Phytopathology* 46, 27-51.

Gooding M J, Dimmock J P R E, France J, Jones S A. 2000. Green leaf area decline of wheat flag leaves: the influence of fungicides and relationships with mean grain weight and grain yield. *Annals of applied Biology* 136, 77-84.

Gooding, M.J. 2007. Influence of foliar diseases and their control by fungicides on grain yield and quality in wheat. In: H.T. Buck et al. (eds.) *Wheat production in stressed environments. Developments in plant breeding* vol. 12, 567-581 Springer

Goodwin, S.B. 2007. Back to basics and beyond: increasing the level of resistance to Septoria tritici blotch in wheat. *Australasian Plant Pathology* 36, 532-538.

Griffin, M.J., Fisher, N. 1985. Laboratory studies on benzimidazole resistance in *Septoria tritici*. *EPPO Bulletin* 15, 505-511.

Griffiths, E. 1993. Iatrogenic effects of pesticides on plant disease – an update and overview. In *Pesticide interactions in crop production. Beneficial and deleterious effects*, ed. Altman J, 269-279.

Guo, J-R., Schnieder, F., Verreet, J-A., 2007. A real-time PCR assay for quantitative and accurate assessment of fungicide effects on *Mycosphaerella graminicola* leaf blotch. J. *Phytopathology* 155, 482-487.

Hall, R., Sutton, J.C., 1998. Relation of weather, crop, and soil variables to the prevalence, incidence, and severity of basal infections of winter wheat in Ontario. *Canadian Journal of Plant Pathology* 20, 69-80.

Hansen, J.G., Secher, B.J.M., Jørgensen, L.N., Welling, B. 1994. Thresholds for control of *Septoria* spp. in winter wheat based on precipitation and growth stage. *Plant Pathology* 43, 183-189.

Hardwick, N.V. 1998. Whither or wither extension plant pathology? British Society for Plant Pathology Presidential ddress 1997. *Plant Pathology* 47, 379-393.

Hardwick N V, Jones D R, Slough J E. 2001. Factors affecting diseases of winter wheat in England and Wales, 1989-1998. *Plant Pathology* 50, 453-462.

Hau, B., de Vallavieille-Pope, C. 1998. Wind-dispersed diseases. In *The Epidemiology of Plant Diseases*, Jones, D.G. (ed.), 323-347.

Helms Jørgensen, J. (ed.). 1991. *Integrated control of cereal mildews: virulence patterns and their change*. Risø National Laboratory, Denmark. ISBN 87-550-1782-7.

Henson, J.M., French, R. 1993. The polymerase chain reaction and plant disease diagnosis. *Annual Review of Phytopathology* 31, 81-109.

Henze, M., Beyer, M., Klink, H., Verreet, J.-A. 2007. Characterizing meteorological scenarios favorable for *Septoria tritici* infections in wheat and estimation of latent periods. *Plant Disease* 91, 1445-1449.

Hermansen, J.E. 1968. Studies on the spread and survival of cereal rust and powdery mildew diseases in Denmark. *Contribution No 87 from the Department of Plant Pathology, The Royal Veterinary and Agricultural College, Copenhagen.*

Hermansen, J.E., Torp, U., Prahm, L.P. 1978. Studies of transport of live spores of cereal mildew and rust fungi across the North Sea. *Grana* 17, 41-46.

HGCA (Home Grown Cereals Authority), 2009. The wheat disease management guide. Spring 2009, 4th edition, pp. 27. www.hgca.com, accessed August 2009.

Hogenboom, N.G. 1993. Economic importance of breeding for disease resistance. In *Durability of disease resistance*, 5-9. Kluwer Dordrecht

Hollins, T.W., Scott, P.R. 1980. Epidemiology of eyespot (*Pseudocercosporella herpotrichoides*) on winter wheat, with particular reference to the period of infection. *Annals of applied Biology* 95, 19-29

Hollins, T.W., Lockley, K.D., Blackman, J.A., Scott, P.R., Bingham, J. 1988. Field performance of Rendezvouz, a wheat cultivar with resistance to eyespot (*Pseudocercosporella herpotrichoides*) derived from *Aegilops ventricosa*. *Plant Pathology* 37, 251-260.

Hollomon, D.W., Wheeler, I.E. 2002. Controlling powdery mildews with chemistry. In *The Powdery Mildews. A Comprehensive Treatise*. Bélanger, R.R., Bushnell, W.R., Dik, A.J., Carver, T.L.W. (eds.), 249-255.

Horsfall, J.G., Cowling, E.B. 1978. Pathometry: The measurement of plant disease. In *Plant Disease – An advanced treatise, vol. II, How disease develops in populations* (eds. Horsfall J.G., Cowling, E.B.), 119-136.

Horsten, J., Fehrmann, H. 1980. Fungicide resistance of *Septoria nodorum* and *Pseudocercosporella herpotrichoides*. I. Effect of fungicide application on the frequency of resistant spores in the field. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 87, 439-453.

Hovmøller, M.S. 1989. Race specific powdery mildew resistance in 31 Northwest European wheat cultivars. *Plant Breeding* 103, 228-234.

Hovmøller, M.S. 2007. Sources of seedling and adult plant resistance to *Puccinia striiformis* f.sp. *tritici* in European wheats. *Plant Breeding* 126, 225-233.

Hovmøller, M.S., Henriksen, K.E. 2008. Application of pathogen surveys, disease nurseries and varietal resistance characteristics in an IPM approach for the control of wheat yellow rust. *European Journal of Plant Pathology* 121, 377-385.

Hovmøller, M.S., Yahyaoui, A.H., Milus, E.A., Justesen, A.F. 2008. Rapid and global spread of two aggressive strains of a wheat rust fungus. *Molecular Ecology* 17, 3818-3826.

Howard, D.D., Chambers, A.Y., Logan, J. 1994. Nitrogen and fungicide effects on yield components and disease severity in wheat. *Journal of Production in Agriculture* 7, 4, 448-454.

Hughes, G., McRoberts, N., Madden, L.V. 2004. Daamen's incidence-severity relationship revisited. *European Journa of Plant Pathology* 110, 759-761.

Hunter, T., Coker, R.R., Royle, D.J. 1999. The teleomorph stage, *Mycosphaerella graminicola*, in epidemics of septoria tritici blotch on winter wheat in the UK. *Plant Pathology* 48, 51-57.

Inuma, T., Khodaparast, S.A., Takamatsu, S. 2007. Multilocus phylogenetic analyses within *Blumeria graminis*, a powdery mildew fungus of cereals. *Molecular Phylogenetics and Evolution* 44, 741-751.

James, W.C., Shih, C.S. 1973. Size and shape of plots for estimating yield losses from cereal foliage diseases. *Experimental Agriculture* 9, 63-71.

James, W.C. 1974. Assessment of plant diseases and losses. *Annual Review of Phytopathology* 12, 27-48.

Jeger, M.J., 2004. Analysis of disease progress as a basis for evaluating disease management practicies. *Annual Review of Phytopathology* 42, 61-82.

Jenkins, J.E.E., Lescar, L. 1980. Use of foliar fungicides on cereals in Western Europe. *Plant Disease* 64, 987-994.

Jenkyn, J.F., Bainbridge, A. 1978. Biology and pathology of cereal powdery mildews. In *The Powdery Mildews*, Spencer, D.M. (ed.), 283-321. Academic Press.

Jiang, G.M., Sun, J.Z., Liu, H.Q., Qu, C.M., Wang, K.J., Guo, R.J., Bai, K.Z., Gao, L.M., Kuang, T.Y., 2003. Changes in the rate of photosynthesis accompanying the yield increase in wheat cultivars released in the past 50 years. *Journal of Plant Research* 116, 347-354. Tokyo.

Jin, Y., Singh, R.P., Ward, R.W., Wanyera, R., Kinyua, M., Njau, P., Fetch, T., Pretorius, Z.A., Yahyaoui, A. 2007. Characterization of seedling infection types and adult plant infection responses of moniogenic Sr gene lines to race TTKS of *Puccinia graminis* f.sp. *tritici*. *Plant Disease* 91, 1096-1099.

Johansson, G., Wiik, L. 1989. Korn och vetemjöldaggspopulationens (*Erysiphe graminis* f. sp. *hordei*) and (*Erysiphe graminis* f. sp. *tritici*) känslighet för fungiciden Tilt 250 EC (propikonazol) i Skåne 1985-1989 (Engl. summary). Nordisk Plantevaernskonference. *Statens Planteavlsforsøg* 149-153.

Johansson, E., Svensson, G. 1998. Variation in bread-making quality: Effects of weather parameters on protein concentration and quality in some Swedish wheat cultivars grown during the period 1975-1996. *Journal of the Science of Food and Agriculture* 78, 109-118.

Johnson, R. 1984. A critical analysis of durable resistance. *Annual Review of Phytopathology* 22, 309-330.

Johnson, R. 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. In *Breeding strategies for resistance to rusts of wheat*, 63-75. CIMMYT

Johnson, R. 1992. Past, present and future oppurtunities in breeding for disease resistance, with examples from wheat. *Euphytica* 63, 3-22.

Johnsson, L. 1992a. Climate factors influencing attack of common bunt (*Tilletia caries* (DC) Tul.) in winter wheat in 1940-1988 in Sweden. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 99, 1, 21-28.

Johnsson, L. 1992b. Dwarf bunt (*Tilletia contraversa* Kühn) in winter wheat in Sweden: relationship to climate (1951-1987), climate, survey results and cultivation measures (1967-1987). *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 99, 3, 256-265.

Johnston, H.W., MacLeod, J.A., Clough, K.S. 1979. Effects of cycocel (CCC) and fungicide sprays on spring wheat grown at three nitrogen levels. *Canadian Journal of Plant Science* 59, 917-929.

Jones, D.R. 1994. Evaluation of fungicides for control of eyespot disease and yield loss nationships in winter wheat. *Plant Pathology* 43, 831-846.

Jones, S.S., Murray, T.D., Allan, R.E. 1995. Use of alien genes for the development of disease resistance in wheat. *Annual Review of Phytopathology* 33, 429-443.

Jönsson, J.Ö. 1978. Race non-specific resistance to *Puccinia striiformis* f.sp. *tritici*. Agri *Hortique Genetica* XXXVI, 13-23.

Jørgensen, L.N., Secher, B.J.M., Nielsen, G.C. 1996. Monitoring diseases of winter wheat on both a field and a national level in Denmark. *Crop Protection* 15, 4, 383-390.

Jørgensen, L.N., Secher, B.J.M., Olesen, J.E., Mortensen, J. 1997. Need for fungicide treatments when varying agricultural parameters. *Aspects of Applied Biology* 50, 285-292.

Jørgensen, L.N., Secher, B.J.M., Hossy, H. 1999. Decision support systems featuring Septoria management. In *Septoria on cereals. A study of pathosystems*, eds. Lucas, J.A., Bowyer, P., Anderson, H.M., 251-262. CABI Publishing ISBN 0 85199 269 2

Kema, G.H.J., Van Silfhout, C.H. 1997. Genetic variation for virulence and resistance in the wheat-*Mycosphaerella graminicola* pathosystem III. Comparative seedling and adult plant experiments. *Phytopathology* 87, 266-272.

Kema, G.H.J., Sayoud, R., Annone, J.G., Van Silfhout, C.H. 1996. Genetic variation for virulence and resistance in wheat-*Mycosphaerella graminicola* pathosystem II. Analysis of interactions between pathogen isolates and host cultivars. *Phytopathology* 86, 213-220.

Kettlewell, P.S., Cook, J.W., Parry, D.W. 2000. Evidence for an osmotic mechanism in the control of powdery mildew disease of wheat by foliar-applied potassium chloride. *European Journal of Plant Pathology* 106, 297-300.

Khoury, W., Kranz, J. 1994. An assessment of linear structural relationships (LISREL) for the analysis of interactions between weather factors in epidemiological studies. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 101, 286-296.

King, J.E. 1976. Relationship between yield loss and severity of yellow rust recorded on a large number of single stems of winter wheat. *Plant Pathology* 25, 172-177.

King, J.E. 1977. Surveys of diseases of winter wheat in England and Wales, 1970-75. *Plant Pathology* 26, 8-20.

King, J.E., Griffin, M.J. 1985. Survey of benomyl resistance in *Pseudocercosporella herpotrichoides* on winter wheat and barley in England and Wales in 1983. *Plant Pathology* 34, 271-283.

King, J.E., Jenkins, J.E.E., Morgan, W.A. 1983. The estimation of yield losses in wheat from severity of infection by *Septoria* species. *Plant Pathology* 32, 239-249.

Kinkel, L.L. 1997. Microbial population dynamics on leaves. *Annual Review of Phytopathology* 35, 327-347.

Klinkowski, M. 1970. Catastrophic plant diseases. *Annual Review of Phytopathology* 8, 37-60.

Knight, S.C., Anthony, V.M., Brady, A.M., Greenland, A.J., Heaney, S.P., Murray, D.C., Powell, K.A., Schulz, M.A., Spinks, C.A., Worthington, P.A., Youle, D. 1997. Rationale and perspectives on the development of fungicides. *Annual Review of Phytopathology* 35, 349-372.

Knight, S., Blake, J., Oxley, S., Parsons, D. 2008. Fungicide doses in sequence and mixtures for winter wheat. *HGCA, project report* No. 439.

Kolmer, J.A. 1991. Phenotypic diversity in two populations of *Puccinia recondita* f. sp. *tritici* in Canada during 1931-1987. *Phytopathology* 81, 311-315.

Kolmer, J.A., Jin, Y., Long, D.L. 2007. Wheat leaf and stem rust in the United States. *Australian Journal of Agricultural Research* 58, 631-638.

Kolmer, J.A., Long, D.L., Hughes, M.E. 2006. Physiologic specialization of *Puccinia triticina* in wheat in the United States in 2004. *Plant Disease* 90, 1219-1224.

Lagerberg, C., Gripwall, E., Wiik, L. 1996. Detection and quantification of seed-borne *Septoria nodorum* in naturally infected grains of wheat with polyclonal ELISA. *Seed Science and Technology* 23, 609-615.

Large, E.C. 1966. Measuring plant disease. *Annual Review of Phytopathology* 4, 9-28.

Large, E.C., Doling, D.A. 1963. Effect of mildew on yield of winter wheat. *Plant Pathology* 12, 128-130.

Larsson, H. 2005. A crop loss model and economic thresholds for the grain aphid, *Sitobion avenae* (F.), in winter wheat in southern Sweden. *Crop Protection* 24, 397-405.

Larsson, S. 1986. New screening methods for drought resistance and cold hardiness in cereals. In *Svalöf 1886-1986. Research and Results in Plant Breeding*, 241-251.

Larsson, S., Magnét, B., Hagman, J. 2001. *Sortval 2001–Stråsäd, trindsäd, oljeväxter, potatis*. SLU Uppsala Sweden. ISBN 91-576-6012-3.

Larsson, S., Hagman, J., Börjesdotter, D. 2003. *Sortval 2003–Stråsäd, trindsäd, oljeväxter, potatis*. SLU Uppsala Sweden. ISBN 91-576-6292-4.

Larsson, S., Hagman, J., Börjesdotter, D. 2005. *Sortval 2005–Stråsäd, trindsäd, oljeväxter, potatis*. SLU Uppsala Sweden. ISBN 91-576-6808-6.

Larsson, S., Hagman, J., Ericson, L. 2008. *Sortval 2008–Stråsäd, trindsäd, oljeväxter, potatis*. SLU Uppsala Sweden. ISBN 978-91-85911-31-8.

Leitch, M.H., Jenkins, P.D. 1995. Influence of nitrogen on the development of Septoria epidemics in winter wheat. *Journal of Agricultural Science*, Cambridge 124, 361-368.

Leath, S., Bowen, K.L. 1989. Effects of powdery mildew, triadimenol seed treatment, and triadimefon foliar sprays on yield of winter wheat in North Carolina. *Phytopathology* 79, 152-155.

Leath, S., Scharen, A.L., Dietzholmes, M.E., Lund, R.E. 1993. Factors associated with global occurrences of Septoria nodorum blotch and Septoria tritici blotch of wheat. *Plant Disease* 77, 1266-1270.

Leijerstam, B. 1961. Physiologic races of cereal rusts in Sweden 1956-1959. *Statens Växtskyddsanstalt. Meddelanden* 12:84, 53-61.

Leijerstam, B. 1972. Race-specific resistance to wheat powdery mildew. A summary of Swedish results. *Statens Växtskyddsanstalt. Meddelanden* 15:146, 273-277.

Leonard, J.M., Watson, C.J.W., Carter, A.H., Hansen, J.L., Zemetra, R.S., et al. 2008. Identification of a candidate gene for the wheat endopeptidase *Ep-D1* locus and two other STS markers linked to the eyespot resistance gene *Pch1*. *Theoretical and Applied Genetics* 116, 261-270.

Leroux, P., Gredt, M. 1997. Evolution of fungicide resistance in the cereal eyespot fungi *Tapesia yallundae* and *Tapesia acuformis* in France. *Pesticide Science* 51, 321-327.

Leroux, P., Albertini, C., Gautier, A., Gredt, M, Walker, A.-S. 2007. Mutations in the CYP51 gene correlated with changes in sensitivity to sterol 14α-demethylation inhibitors in field isolates of *Mycosphaerella graminicola*. *Pesticide Management Science* 63, 688-698.

Lévesque, C.A., Rahe, J.E. 1992. Herbicide interactions with fungal root pathogens, with special reference to glyphosate. *Annual Review of Phytopathology* 30, 579-602.

Li, H.J., Arterburn, M., Jones, S.S., Murray, T.D. 2004. A new source of resistance to *Tapesia yallundae* associated with a homoeologous group 4 chromosome in *Thinopyrum ponticum*. *Phytopathology* 94, 932-937.

Li, H.J., Arterburn, M., Jones, S.S., Murray, T.D. 2005. Resistance to eyespot of wheat, caused by *Tapesia yallundae*, derived from *Thinopyrum intermedium* homoeologous group 4 chromosome. *Theoretical and Appplied Genetics* 111, 932-940.

Limpert, E., Finckh, M.R., Wolfe, M.S. (eds.) 1996. *COST 817. Integrated control of cereal diseases and rusts: Towards coordination of research across Europe*. EUR 16884 EN. ISBN 92-827-7171.

Lind, V. 1999. Variation of resistance to *Pseudocercosporella herpotrichoides* (Fron) Deighton in wheat genotypes carrying the gene *Pch-1*. *Plant Breeding* 118, 281-287.

Line, R.F. 2002. Stripe rust of wheat and barley in North America: A retrospective historical review. *Annual Review of Phytopathology* 40, 75-118.

Lipps, P.E., Madden, L.V. 1989. Effect of fungicide application timing on control of powdery mildew and grain yield of winter wheat. *Plant Disease* 73, 991-994.

Ljungars, A. 2001. Sortförsök i höstvete. In *Skåneförsök 2000. Jordbruksförsöken i Skåne län. Meddelande* 67, 16-28.

Lockley, D., Clark, W.S. 2005. Fungicide dose-response trials in wheat: the basis for choosing 'appropriate' dose. *HGCA, project report* No. 373.

Lockley, D., Clark, B., Oxley, S., Self, M., Ainsley, A., Dyer, C., Paveley, N., Dunne, B. 2008. Appropriate doses network: up-to-date information on fungicide performance for wheat growers. *HGCA, project report* No. 444.

Love, C.S., Bruehl, G.W. 1987. Effect of soil pH on cephalosporium stripe in wheat. *Plant Disease* 71, 727-731.

Lovell, D.J., Parker, S.R., Hunter, T., Royle, D.J., Coker, R.R. 1997. Influence of crop growth and structure on the risk of epidemics by *Mycosphaerella graminicola* (*Septoria tritici*) in winter wheat. *Plant Pathology* 46, 126-138.

Lovell, D.J., Parker, S.R., Hunter, T., Welham, S.J., Nichols, A.R. 2004. Position of inoculum in the canopy affects the risk of septoria tritici blotch epidemics in winter wheat. *Plant Pathology* 53, 11-21.

Loyce, C., Meynard, J.M., Bouchard, C., Rolland, B. et al. 2008. Interaction between cultivar and crop management effects on winter wheat diseases, lodging, and yield. *Crop Protection* 27, 1131-1142.

Lucas, J. 2003. Resistance to QoI fungicides: Implications for cereal disease management in Europe. *Pesticide Outlook* – December, 268-270.

Lucas, J.A., Dyer, P.S., Murray, T.D. 2000. Pathogenicity, host specificity, and population biology of *Tapesia* spp., causal agents of eyespot disease of cereals. *Advances in Botanical Research* 33, 225-258.

Lundin, P. 1973. Resistance breeding to cereal rust diseases in Sweden. *Sveriges Utsädesförenings Tidskrift* 83, 66-69.

Lundin, P. 1997. Rost- och mjöldaggsresistens hos stråsäd. In Den svenska växtförädlingens historia. *Kungl. Skogs- och Lantbruksakademien. Skogs- och lantbrukshistoriska meddelanden* 20, 145-154.

Mac Key, J. 1993. Demonstration of genetic gain from Swedish cereal breeding. *Sveriges Utsädesförenings Tidskrift* 1, 33-43.

Magboul, A.M., Geng, S., Gilchrist, D.G., Jackson, L.F. 1992. Environmental influence on the infection of wheat by *Mycosphaerella graminicola*. *Phytopathology* 82, 1407-1413.

Mann, R.L., Kettlewell, P.S., Jenkinson, P. 2004. Effect of foliar-applied potassium chloride on septoria leaf blotch of winter wheat. *Plant Pathology* 53, 653-659.

Marasas, C.N., Smale, M., Singh, R.P. 2003. The economic impact of productivity maintenance research: breeding for leaf rust resistance in modern wheat. *Agricultural Economics* 29, 253-263.

Martin, R.R, James, D., Lévesque, C.A. 2000. Impacts of molecular diagnostic technologies on plant disease management. *Annual Review of Phytopathology* 38, 207-239.

Mavroeidi, V.I., Shaw, M.W. 2005. Sensitivity distributions and cross-resistance patterns of Mycosphaerella graminicola to fluquinconazole, prochloraz and azoxystrobin over a period of 9 years. *Crop Protection* 24, 259-266.

McCallum, B.D., Seto-Goh, P. 2008. Physiologic specialization of *Puccinia triticina* in Canada in 2005. *Canadian Journal of Plant Pathology* 30, 124-132.

McCartney, C., Mercer, P.C., Cooke, L.R., Fraaije, B.A. 2007. Effects of a strobilurin-based spray programme on disease control, green leaf area, yield and development of fungicide resistance in *Mycosphaerella graminicola* in Northern Ireland. *Crop Protection* 26, 1272-1280.

McDonald, W.C., Martens, J.W., Nielsen, J., Green, G.J., Samborski, D.J. et al. 1971. Losses from cereal diseases and value of disease resistance in Manitoba and eastern and northern Saskatchewan in 1970. *Canadian Plant Disease Survey* 51, 105-110.

McDonald, B.A., Linde, C. 2002. Pathogen population genetics, evolutionary potential, and durable resistance. *Annual Review of Phytopathology* 40, 349-379.

McIntosh, R.A. 2007. From Farrer to the Australian cereal rust control program. *Australian Journal of Agricultural Research* 58, 550-557.

McIntosh, R.A., Brown, G.N. 1997. Anticipatory breeding for resistance to rust diseases in wheat. *Annual Review of Phytopathology* 35, 311-326

McRoberts, N., Hughes, G., Madden, L.V. 2003. The theoretical basis and practical application of relationships between different disease intensity measurements in plants. *Annals of applied Biology* 142, 191-211.

McVey, D.V., Long, D.L., Roberts, J.J. 2002. Races of *Puccinia graminis* in the United States during 1997 and 1998. *Plant Disease* 86, 568-572.

Mercer, P.C., Ruddock, A. 2004. Surveys of cereal diseases in Northern Ireland, 1976 to 2000. *Irish Journal of Agricultural and Food Research* 43, 85-101.

Mercer, P.C., Ruddock, A. 2005. Disease management of winter wheat with reduced doses of fungicides in Northern Ireland. *Crop Protection* 24, 221-228.

Mesterházy, A., Bartos, P., Goyeau, H., Niks, R.E. et al. 2000. European virulence survey for leaf rust in wheat. *Agronomie* 20, 793-804.

Metcalfe, R.J., Shaw, M.W., Russel, P.E. 2000. The effect of dose and mobility on the strength of selection for DMI fungicide resistance in inoculated field experiments. *Plant Pathology* 49, 546-557.

Miller, S.A., Beed, F.D., Harmon, C.L. 2009. Plant disease diagnostic capabilities and networks. *Annual Review of Phytopathology* 47, 15-38.

Milne, A., Paveley, N., Audsley, E., Parsons, D. 2007. A model of the effect of fungicides on disease-induced yield loss, for use in wheat disease management decision support systems. *Annals of Applied Biology* 151, 113-125.

Milus, E.A., Kristensen, K., Hovmøller, M.S. 2009. Evidence for increased aggressiveness in a recent widespread strain of *Puccinia striiformis* f. sp. *tritici* causing stripe rust of wheat. *Phytopathology* 99, 89-94.

Morell M. 2001. *Det svenska jordbrukets historia. Jordbruket i industrisamhället 1870-1945.* Natur och Kultur, LTs förlag.

Morris, C.E., Sands, D.C. 2006. The breeeder's dilemma–yield or nutrition? *Nature Biotechnology* 24, 1078-1080.

Moschinin, R.C., Pérez, B.A. 1999. Predicting wheat leaf rust severity using planting date, genetic resistance, and weather variables. *Plant Disease* 83, 381-384.

Mundt, C.C., Brophy, L.S., Schmitt, M.S. 1995a. Choosing crop cultivars and cultivar mixtures under low versus high disease pressure: A case study with wheat. *Crop Protection* 14, 509-515.

Mundt, C.C., Brophy, L.S., Schmitt, M.S. 1995b. Disease severity and yield of pure-line wheat cultivars and mixtures in the presence of eyespot, yellow rust, and their combination. *Plant Pathology* 44, 173-182.

Mundy, E.J. 1973. The effect of yellow rust and its control of the yield of Joss Cambier winter wheat. *Plant Pathology* 22, 171-176.

Munkvold, G.P. 2009. Seed pathology progress in academia and industry. *Annual Review of Phytopathology* 47, 285-311.

Muranty, H., Jahier, J., Tanguy, A.-M., Worland, A.J., Law, C. 2002. Inheritance of resistance of wheat to eyespot at the adult stage. *Plant Breeding* 121, 536-538.

Murray, G.M., Martin, R.H., Cullis, B.R. 1990. Relationship of the severity of septoria tritici blotch of wheat to sowing time, rainfall at heading and average susceptibility of wheat cultivars in the area. *Australian Journal of Agricultural Research* 41, 307-315.

Murray, T.D., de la Pena, R.C., Yildirim, A., Jones, S.S. 1994. A new source of resistance to *Pseudocercosporella herpotrichiodes*, cause of eyespot disease of wheat, located on chromosome 4V of *Dasypyrum villosum*. *Plant Breeding* 113, 281-286.

Murray, T.D. 1996. Resistance to benzimidazole fungicides in the cereal eyespot pathogen, *Pseudocercosporella herpotrichiodes*, in the Pacific Northwest 1984 to 1990. *Plant Disease* 80, 19-23.

Neumann, S., Paveley, N.D., Beed, F.D., Sylvester-Bradley, R. 2004. Nitrogen per unit leaf area affects the upper asymptote of *Puccinia striiformis* f.sp. *tritici* epidemics in winter wheat. *Plant Pathology* 53, 725-732.

NIAB (National Institute of Agricultural Botany). 2009. www.niab.com, accessed Aug 2009.

Nicolas, H. 2004. Using remote sensing to determine of the date of a fungicide application on winter wheat. *Crop Protection* 23, 853-863.

Nilsson-Ehle, H. 1904. On some of our most important plant diseases and their economic importance for agriculture. *Sveriges Utsädesförenings Tidskrift* 14, 163-174.

Nilsson H-E. 1995. Remote sensing and image analysis in plant pathology. *Canadian Journal of Plant Pathology* 17, 154-166.

Nordgen 2009. NordGen, www.nordgen.org, accessed August 2009.

Nutter, F.W., Teng, P.S., Royer, M.H. 1993. Terms and concepts for yield, crop loss, and disease thresholds. *Plant Disease* 77, 211-215.

Oerke, E-C., Dehne, H-W., Schönbeck, F., Weber, A. 1994. *Crop production and crop protection. Estimated losses in major food and cash crops*. Elsevier Science BV The Netherlands.

Olesen, J.E., Jørgensen, L.N., Petersen, J., Mortensen, J.V. 2003. Effects of rate and timing of nitrogen fertilizer on disease control by fungicides in winter wheat. 1. Grain yield and foliar disease control. *Journal of Agricultural Science* 140, 1-13.

Olofsson, S. 1993. Influence of preceding crop and crop residue on stand and yield of winter wheat (*Triticum aestivum* L.), in different tillage systems, including zero tillage. *Crop Production Science 18. SLU*, Uppsala, Sweden.

Olvång, H. 1987. Investigations of resistance to fungicides in some plant pathogen – fungicide systems in Sweden. Plant protection reports. *Dissertations* 16. SLU, Uppsala.

Olvång, H. 1988. Sensitivity of *Drechslera teres* and *Septoria nodorum* to sterolbiosynthesis inhibitors. *Netherlands Journal of Plant Pathology* 94, 57-68.

Olvång, H. 1990. Stråknäckarsvampen - möjlighet till prognos, resultat från försöksverksamheten. *31:a svenska växtskyddskonferensen. Skadedjur och växtsjukdomar*, 255-260.

Onstad, D.W., Rabbinge, R. 1985. Dynamic programming and the computation of economic injury levels for crop disease control. *Agricultural Systems* 18, 207-226.

Ortiz, R., Trethowan, R., Ortiz Ferrara, G., Iwanaga, M., Dodds, J.H., Crouch, J.H., Crossa, J., Braun, H.-J. 2007. High yield potential, shuttle breeding, genetic diversity, and a new international wheat improvement strategy. *Euphytica* 157, 365-384.

Oste, B., Huguerot, G., Delos, M., Freydier, M., Henaff, G.L. et al. 2000. Bilan sanitaire de la campagne 1998/1999. *Phytoma* 523, 12-14.

Østergård, H. (chairperson). 2000. Cost 817. *Agronomie* 20, 713-787.

Palmer, C.-L., Skinner, W. 2002. *Mycosphaerella graminicola*: latent infection, crop devastation and genomics. *Molecular Plant Pathology* 3, 63-70.

Papastamati, K., van den Bosch, F. 2007. The sensitivity of the epidemic growth rate to weather variables, with an application to yellow rust on wheat. *Phytopathology* 97, 202-210.

Park, R.F. 2008. Breeding cereals for rust resistance in Australia. *Plant Pathology* 57, 591-602.

Parks, R., Carbone, I., Murphy, J.P., Cowger, C. 2009. Population genetic analysis of an eastern U.S. wheat powdery mildew population reveals geographic subdivision and recent common ancestry with U.K. and Israeli populations. *Phytopathology* 99, 840-849.

Parker, S.R., Lovell, D.J., Royle, D.J., Paveley, N.D. 1999. Analysing epidemics of *Septoria tritici* for improved estimates of disease risk. In *Septoria on cereals. A study of pathosystems*, eds. Lucas JA, Bowyer P, Anderson HM, 96-107. CABI Publishing.

Parsons, D.J., Te Beest, D. 2004. Optimising fungicide applications on winter wheat using genetic algorithms. *Biosystems Engineering* 88, 401-410.

Paveley, N.D. 1999. Integrating *Septoria* risk variables. In *Septoria on cereals. A study of pathosystems*, eds. Lucas JA, Bowyer P, Anderson HM, 230-250. CABI Publishing.

Paveley, N.D., Lockley, D., Vaughan, T.B., Thomas, J., Schmidt, K. 2000. Predicting effective fungicide doses through observation of leaf emergence. *Plant Pathology* 49, 748-766.

Paveley, N.D., Sylvester-Bradley, R., Scott, R.K., Craigon, J., Day, W. 2001. Steps in predicting the relationship of yield on fungicide dose. *Phytopathology* 91, 708-716.

Paveley, N.D., Thomas, J., Vaughan, T.B., Havis, N.D., Jones, D.R. 2003. Predicting effective doses for the joint action of two fungicide applications. *Plant Pathology* 52, 638-647.

Pedigo, L.P., Hutchins, S.C., Higley, L.G. 1986. Economic injury levels in theory and practice. *Annual Review of Entomology* 31, 341-368.

Pedersen, E.A., Hughes, G.R. 1992. The effect of crop rotation on development of the septoria disease complex on spring wheat in Saskatchewan. *Canadian Journal of Plant Pathology* 14, 152-158.

Pedersen, W.L., Leath, S. 1988. Pyramiding major genes for resistance to maintain residual effects. *Annual Review of Phytopathology* 26, 369-378.

Persson, G., Mattsson, B., Kristiansson, B. 1986. Breeding self fertilizers. In *Svalöf 1886-1986. Research and Results in Plant Breeding*, 62-69.

Pietravalle, S., Shaw, M.W., Parker, S.R., van den Bosch, F. 2003. Modeling of relationships between weather and *Septoria tritici* epidemics on winter wheat: A critical approach. *Phytopathology* 93, 1329-1339.

Polley, R.W., Thomas, M.R. 1991. Surveys of diseases of winter wheat in England and Wales, 1976-1988. *Annals of Applied Biology* 119, 1-20.

Priestley, R.H. 1981. Choice and deployment of resistant cultivars for cereal disease control. In: *Strategies for the control of cereal disease*, Jenkyn, J.F. and Plumb, R.T. eds., 65-72

Priestley, R.H., Bayles, R.A. 1988. The contribution and value of resistant cultivars to disease control in cereals. In: *Control of plant diseases: Costs and benefits*, Clifford BC and Lester E eds., 53-65.

Pring, D.R., Lonsdale, D.M. 1989. Cytoplasmic male sterility and maternal inheritance of disease susceptibility in maize. *Annual Review of Phytopathology* 27, 483-502.

Rao, K.V.S., Yang, X.B., Berggren, G.T., Snow, J.P. 1989. A multiple regression model to estimate the contributions of leaves and the effects of leaf rust on yield of winter wheat. *Phytopathology* 79, 1233-1238.

Ray, R.V., Jenkinson, P., Edwards, S.G. 2004. Effects of fungicides on eyespot, caused predominantly by *Oculimacula acuformis*, and yield of early-drilled winter wheat. *Crop Protection* 23, 1199-1207.

Reif, J.C., Zhang, P., Dreisigacker, S., Warburton, M.L., van Ginckel, M., Hoisington, D., Bohn, M., Melchinger, A.E. 2005. Wheat genetic diversity trends during domestication and breeding. *Theoretical and Applied Genetics* 110, 859-864.

Reinink, K. 1986. Experimental verification and development of EPIPRE, a supervised disease and pest management system for wheat. *Netherlands Journal of Plant Pathology* 92, 3-14

Richardson, M.J., Whittle, A.M., Jacks, M. 1976. Yield-loss relationships in cereals. *Plant Pathology* 25, 21-30.

Rodgers-Gray, B.S., Shaw, M.W. 2000. Substantial reductions in winter wheat diseases caused by addition of straw but not manure to soil. *Plant Pathology* 49, 590-599.

Rodgers-Gray, B.S., Shaw, M.W. 2004. Effect of straw and silicon amendments on some foliar and stem-base diseases in pot-grown winter wheat. *Plant Pathology* 53, 733-740.

Roelfs, A.P. 1988. Genetic control of phenotypes in wheat stem rust. *Annual Review of Phytopathology* 26, 351-367.

Roelfs, A.P. 1989. Epidemiology of the cereal rusts in North America. *Canadian Journal of Plant Pathology* 11, 86-90.

Rosielle, A.A. 1972. Sources of resistance in wheat to speckled leaf blotch caused by *Septoria tritici*. *Euphytica* 21, 152-161.

Rossi, V., Giosuè, S. 2003. A dynamic simulation model for powdery mildew epidemics on winter wheat. *EPPO Bulletin* 33, 389-396.

Rossi, V., Racca, P., Giosuè, S., Pancaldi, D., Alberti, I. 1997. A simulation model for the development of brown rust epidemics in winter wheat. *European Journal of Plant Pathology* 103, 453-465.

Rotem, J. 1978. Climatic and weather influences on epidemics. In: *Plant Disease: An advanced treatise*, ed. JG Horsfall, EB Cowling, 2, 317-337.

Royle, D.J., Lovell, D.J., Coakley, S.M., Shaner, G. 1993. Predicting the effects of climate change on Septoria tritici in winter wheat. *Abstracts of the 6th International Congress of Plant Pathology, Montreal*, p. 97.

Ruske, R.E., Gooding, M.J., Dobraszczyk, B.J. 2004. Effects of triazole and strobilurin fungicide programmes, with and without late-season nitrogen fertiliser, on the baking quality of Malacca winter wheat. *Journal of Cereal Sciience* 40, 1-8.

Sanderson, F.R. 1972. A *Mycosphaerella* species as the ascogenous state of *Septoria tritici* Rob. and Desm. *New Zealand Journal of Botany* 10, 707-710

Sanderson, F.R. 1976. *Mycosphaerella graminicola* (Fuckel) Sanderson comb. Nov., the ascogenous state of *Septoria tritici* Rob. apud Desm. *New Zealand Journal of Botany* 14, 359-360.

Savary, S., Teng, P.S., Willocquet, L., Nutter, Jr. F.W. 2006. Quantification and modelling of crop losses: a review of purposes. *Annual Review of Phytopathology* 44, 89-112.

Schepers, H.T.A.M., Bouma, E., Frahm, J., Volk, T., Secher, B.J.M. 1996. Control of fungal diseases in winter wheat with appropriate dose rates and weather-based decision support systems. *OEPP/EPPO Bulletin* 26, 623-630.

Schöfl, U.A., Zinkernagel, V. 1997. A test method based on microscopic assessments to determine curative and protectant fungicide properties against *Septoria tritici*. *Plant Pathology* 46, 545-556.

Scott, P.R., Hollins, T.W. 1974. Effects of eyespot on the yield of winter wheat. *Annals of Applied Biology* 78, 269-279.

Scott, P.R., Hollins, T.W. 1978. Prediction of yield loss due to eyespot in winter wheat. *Plant Pathology* 27, 125-131.

Scott, P.R., Sanderson, F.R., Benedikz, P.W. 1988. Occurrence of *Mycosphaerella graminicola*, teleomorph of *Septoria tritici*, on wheat debris in the UK. *Plant Pathology* 37, 285-290.

Seck, M., Roelfs, A.P., Teng, P.S. 1988. Effect of leaf rust (*Puccinia recondita tritici*) on yield of four isogenic wheat lines. *Crop Protection* 7, 39-42.

Seem, R.C. 1984. Disease incidence and severity relationships. *Annual Review of Phytopathology* 22, 133-150.

Shaner, G., Finney, R.E. 1976. Weather and epidemics of Septoria leaf blotch of wheat. *Phytopathology* 66, 781-785.

Shaw, M.W., Royle, D.J. 1986. Saving Septoria fungicide sprays: the use of disease forecast. 1986 *British Crop Protection Conference - Pests and Diseases* vol. 3, 1193-1200.

Shaw, M.W. 1987. Assessment of upward movement of rain splash using a fluorescent tracer method and its application to the epidemiology of cereal pathogens. *Plant Pathology* 36, 201-213.

Shaw, M.W., Royle, D.J. 1987. Spatial distributions of *Septoria nodorum* and *S. tritici* within crops of winter wheat. *Plant Pathology* 36, 84-94.

Shaw, M.W., Royle, D.J. 1989. Estimation and validation of a function describing the rate at which *Mycosphaerella graminicola* causes yield loss in winter wheat. *Annals of applied Biology* 115, 425-442.

Shaw, M.W., Royle, D.J. 1993. Factors determining the severity of *Mycosphaerella graminicola* (*Septoria tritici*) on winter wheat in the UK. *Plant Pathology 42*, 882-899.

Shaw, M.W., Bearchell, S.J., Fitt, B.D.L., Fraaije, B.A. 2008. Long-term relationships between environment and abundance in wheat of *Phaeosphaeria nodorum* and *Mycosphaerella graminicola*. *New Phytologist* 177, 229-238.

Shipton, P.J. 1977. Monoculture and soilborne plant pathogens. *Annual Review of Phytopathology* 15, 387-407.

Siegfried, W., Viret, O., Huber, B., Wohlhauser, R. 2007. Dosage of plant protection products adapted to leaf area index in viticulture. *Crop Protection* 26, 73-82.

Sieling, K., Ubben, K., Christen, O. 2007. Effects of preceding crop, sowing date, N fertilization and fluquinconazole seed treatment on wheat growth, grain yield and take-all. *Journal of Plant Disease Protection* 5, 213-220.

Sierotzki, H., Wullschleger, J., Gisi, U. 2000. Point mutation in cytochrome b gene conferring resistance to strobilurin fungicides in *Erysiphe graminis* f. sp. *tritici* field isolates. *Pesticide Biochemistry and Physiology* 68, 107-112.

Sigvald, R. 1984. Växtskyddsåret 1983 - Jordbruk. *Växtskyddsnotiser* 48:1, 2-4.

Sim IV, T., Willis, W.G., Eversmeyer, M.G. 1988. Kansas Plant Disease Survey. *Plant Disease* 72 (10), 832-836.

Simón, M.R., Cordo, C.A., Perello, A.E., Struik, P.C. 2003. Influence of nitrogen supply on the susceptibility of wheat to *Septoria tritici*. *Journal of Phytopathology* 151, 283-289.

Simón, M.R., Perello, A.E., Cordo, C.A., Larrán, S., van der Putten, P.E.L, Struik, P.C. 2005. Association between septoria tritici blotch, plant height, and heading date in wheat. *Agronomy Journal* 97, 1072-1081.

SJV, 2009. www.sjv.se, accessed March 2009.

Smale, M., Singh, R.P., Sayre, K., Pingali, P., Rajaram, S., Dubin, H.J. 1998. Estimating the economic impact of breeding nonspecific resistance to leaf rust in modern bread wheats. *Plant Disease* 82, 1055-1061.

Smeets, E., Geypens, M. 1995. The use of meteorological and field specific parameters to predict the evolution of pests and diseases in winter wheat: An analysis of the EPIPRE-database. *Mededelingen Faculteit Landbouwkundige en Toegepaste Biologische Wetenschappen Universiteit Gent* 60 (3A), 743-749.

SMHI, 2009. www.smhi.se, accessed February 2009.

Smiley, R.W., Cook, R.J. 1973. Relationship between take all of wheat and rhizosphere pH in soils fertilized with ammonium vs. nitrate-nitrogen. *Phytopathology* 63, 882-890.

Smith, G.P., Gooding, M.J. 1999. Models of wheat grain quality considering climate, cultivar and nitrogen effects. *Agricultural and Forest Meteorology* 94, 159-170.

Staub, T. 1991. Fungicide resistance: Practical experience with antiresistance strategies and the role of integrated use. *Annual Review of Phytopathology* 29, 421-442.

Stevens, D.B., Turner, J.A., Paveley, N.D. 1997. Exploiting variety resistance to rationalise fungicide inputs – theory and practice. *Aspects of Applied Biology* 50, Optimising Cereal Inputs; Its Scientific Basis, Part 2: Crop Protection and Systems, 279-284.

Strange, R.N., Scott, P.R. 2005. Plant disease: A threat to global food security. *Annual Review of Phytopathology* 43, 83-116.

Stynes, B.A., Veitch, L.G. 1983. A synoptic approach for crop loss assessment used to study wheat. VI. The pathogen data and their relationship to soil and cultural practice data. *Australian Journal of Agricultural Research* 34, 167-181.

Suzuki, H. 1975. Meteorological factors in the epidemiology of rice blast. *Annual Review of Phytopathology* 13, 239-256.

Svensson, G. 1981. Varietal and environmental effects on wheat milling quality. *Agri Hortique Genetica* XXXIX, 1-103.

Svensson, G. 1997. Veteförädlingen i Sverige. In *Den svenska växtförädlingens historia. Kungl. Skogs- och Lantbruksakademien. Skogs- och lantbrukshistoriska meddelanden* 20, 169-176.

Sweeney, D.W., Granade, G.V., Eversmeyer, M.G., Whitney, D.A. 2000. Phosphorus, potassium, chloride, and fungicide effects on wheat yield and leaf rust severity. *Journal of Plant Nutrition* 23, 1267-1281.

Tapke, V.F. 1951. Influence of preinoculation environment on the infection of barley and wheat by powdery mildew. *Phytopathology* 41, 622-632.

Tavella, C.M. 1978. Date of heading and plant height of wheat varieties, as related to Septoria leaf blotch damage. *Euphytica* 27, 577-580.

Te Beest, D.E., Paveley, N.D., Shaw, M.W., van den Bosch, F. 2008. Disease-weather relationships for powdery mildew and yellow rust on winter wheat. *Phytopathology* 98, 609-617.

Teng, P.S., Krupa, S.V. (eds). 1980. Assessment of losses which constrain production and crop improvement in agriculture and forestry. *Proceedings of EC Stakman Commemorative Symposium. Miscellaneous Publications* 7, University of Minnesota.

Teng, P.S., Blackie, M.J., Close, R.C. 1977. A simulation analysis of crop yield loss due to rust disease. *Agricultural Systems* 2, 189-198.

The Economist. 2005. Ears of plenty. The story of wheat. www.economist.com, accessed August 2009.

Thomas, M.R., Cook, R.J., King, J.E. 1989. Factors affecting development of *Septoria tritici* in winter wheat and its effect on yield. *Plant Pathology* 38, 246-257.

Thygesen, K., Jørgensen, L.N., Skov Jensen, K., Munk, L. 2009. Spatial and temporal impact of fungicide spry strategies on fungiocide sensitivity of *Mycosphaerella graminicola* in winter wheat. *European Journal of Plant Pathology* 123, 435-447.

Tigerstedt, P.M.A (ed.). 1997. *Adaption in plant breeding. Developments in Plant Breeding.* Kluwer Academic Publishers.

Tompkins, D.K., Fowler, D.B., Wright, A.T., 1993. Influence of agronomic practices on canopy microclimate and septoria development in no-till winter wheat produced in the Parkland region of Saskatchewan. *Canadian Journal of Plant Science* 73, 331-344.

Torriani, S.F.F, Brunner, P.C., McDonald, B.A., Sierotzki, H. 2009. QoI resistance emerged independently at least 4 times in European populations of *Mycosphaerella graminicola. Pesticide Management Science* 65, 155-162.

Tottman, D.R., Broad, H. 1987. The decimal code for the growth stages of cereals, with illustrations. *Annals of Applied Biology* 110, 441-454.

Trottet, M. 2001. La résistance des variétés aux maladies: La septoriose. *Perspective Agricole* 268, p. 44.

Turner, A.S., Nicholson, P., Edwards, S.G., Bateman, G.L., Morgan, L.W., Todd, A.D., Parry, D.W., Marshall, J., Nuttall, M. 2001. Evaluation of diagnostic and quantitative PCR for the identification and severity assessment of eyespot in winter wheat. *Plant Pathology* 50, 463-469.

van den Berg, F., van den Bosch, F. 2007. The elasticity of the epidemic growth rate to observed weather patterns with an application to yellow rust. *Phytopathology* 97, 1512-1518.

van der Planck, J.E. 1963. *Plant diseases: epidemics and control.* Academic Press.

Vanderplank, J.E. 1982. *Host-pathogen interactions in plant disease.* Academic Press.

Vanderplank, J.E. 1984. *Disease resistance in plants.* Second edition. Academic Press.

Van Ginkel, M., Scharen, A.L. 1988. Host-pathogen relationships of wheat and *Septoria tritici*. *Phytopathology* 78, 762-766.

Van Loon, L.C., Bakker, P.A.H.M., Pieterse, C.M.J. 1998. Systemic resistance induced by rhizosphere bacteria. *Annual Review of Phytopathology* 36, 453-483.

Varga, B., Svecnjak, Z., Macesic, D., Uher, D. 2005. Winter wheat cultivar responses to fungicide application are affected by nitrogen fertilization rate. *Journal of Agronomy and Crop Science* 191, 130-137.

Vechet, L. 1992. Influence of some weather factors on epidemics of Puccinia striiformis f. sp. tritici. *Cereal Rusts and Powdery mildews Bulletin* 20, (1-2), 38-42.

Verreet, J.-A. 1995. Principles of integrated pest management. The IPM wheat model. *Pflanzenschutz-Nachrichten Bayer* 48, pp. 307.

Verreet, J.A., Klink, H., Hoffmann, G.M. 2000. Regional monitoring for disease prediction and optimization of plant protection measures: The IPM wheat model. *Plant Disease* 84, 816-826.

Waern, P. 1989. Vetets brunrost – En skadegörare av betydelse i Sverige? (Engl. summary). *30:e svenska växtskyddskonferensen*, 134-143. SLU Uppsala.

Walker, P.T. 1983. Crop losses: The need to quantify the effects of pests, diseases and weeds on agricultural production. *Agriculture, Ecosystems and Environment* 9, 119-158.

Walklate, P.J., McCartney, H.A., Fitt, B.D.L. 1989. Vertical dispersal of plant pathogens by splashing. Part II: experimental study of the relationship between raindrop size and the maximum splash height. Plant Pathology 38, 64-70.

Walklate, P.J., Cross, J.V., Richardson, G.M., Baker, D.E., Murray, R.A. 2003. A generic method of pesticide dose expression: Application to broadcast spraying of apple trees. *Annals of applied Biology* 143, 11-23.

Walters, D.R., Bingham, I.J. 2007. Influence of nutrition on disease development caused by fungal pathogens: implications for plant disease control. *Annals of Applied Biology* 151, 307-324.

Wang, J., Pawelzik, E., Weinert, J., Zhao, Q., Wolf, G.A. 2004. Effect of fungicide treatment on the quality of wheat flour and breadmaking. *Journal of Agricultural and Food Chemistry* 52, 7593-7600.

Wang, J., Pawelzik, E., Weinert, J, Zhao, Q, Wolf, G.A., 2008. Factors influencing falling number in winter wheat. *European Food Research and Technology* 226, 1365-1371.

Welch, R.M., Graham, R.D. 1999. A new paradigm for world agriculture: meeting human needs: Productive, sustainable, nutritious. *Field Crops Research* 60, 1-10.

Welinder, S., Pedersen, E.A., Widgren, M. 1998. *Det svenska jordbrukets historia. Jordbrukets första fem tusen år 4000 f.Kr-1000eKr.* Natur och Kultur, LTs förlag.

Wellings, C.R. 2007. *Puccinia striiformis* in Australia: a review of the incursion, evolution, and adaption of stripe rust in the period 1979-2006. *Australian Journal of Agricultural Research* 58, 567-575.

Wiese, J., Bagy, M.M.K., Schubert, S. 2003. Soil properties, but not plant nutrients (N, P, K) interact with chemically induced resistance against powdery mildew in barley. *Journal of Plant Nutrition Science* 166, 379-384.

Wiese, M.V. 1977. *Compendium of wheat diseases.* The American Phytopathological Society. USA. ISBN 0-89054-023-3.

Wiese, R.J., Veseth, R.J. 1991. *Wheat health management*, 152 pp. APS Press, USA.

Wiik, L. 1981. 1980 års försök mot mjöldagg och brunfläcksjuka. *Växtskyddsrapporter. Jordbruk* 15, 74-82.

Wiik, L. 1982. Preparatval vid mjöldaggsbekämpning. *Växtskyddsrapporter. Jordbruk* 20, 118-126

Wiik, L. 1985. Bekämpning av svampsjukdomar i höstvete. I. Fungicidsprutning kan både öka och minska kärnskördens smittograd. (Engl. summary). *Växtskyddsnotiser* 49, 62-69.

Wiik L. 1986. Stråbassjukdomar i höstvete – en analys av olika variabler i 30 odlingssystemförsök i Skåne 1983-1985. Nordisk plantevernkonferanse 1986. *Aktuelt fra Statens fagtjenste for landbruket* 8, 59-70.

Wiik, L. 1990. Rost i stråsäd i SJFD. *Meddelande från södra jordbruksförsöksdistriktet* 36, 9:1-9:16. SLU Alnarp.

Wiik, L. 1991. Mildew populations of barley and wheat in Sweden. In *Integrated control of cereal mildews: Virulence patterns and their change* (Helms Jørgensen, J. ed.), 33-37. Risø National Laboratory, Denmark.

Wiik, L. 1993. Väderleken och *Septoria* spp.: Sambandet mellan några klimatparametrar och skördeförlusten orsakad av *Septoria* spp. (Engl. summary). *34:e svenska växtskyddskonferensen. Uppsala 27-28 januari 1993. Skadedjur och växtsjukdomar*, 85-90.

Wiik L, Olofsson B, Johnsson L, Olvång H. 1995. Sprutning mot skadesvampar i stråsäd med fungicider 1976-1992. *SLU institutionen för växtskyddsvetenskap Alnarp Rapport* 3, pp. 115.

Wiik, L., Ewaldz, T., Pålsson, L. 1996. Fungicidbehandling i höstsäd 1996: Preparatprovning. SLU. *Meddelande från södra jordbruksförsöksdistriktet* 47, 28A:1-28A:14.

Wolfe, M.S., Schwarzbach, E. 1975. The use of virulence analysis in cereal mildews. *Phytopathologische Zeitschrift* 82, 297-307.

Wolfe, M.S., Slater, S.E., Minchin, P.N. 1981. Mildew of barley. *U.K. Cereal Pathogen Survey. 1980 Annual Report*, 42-56.

Wolfe, M.S., Limpert, E. (eds.). 1987. Integrated control of cereal mildews: Monitoring the pathogen. *Advances in Agricultural Biotechnology*. ISBN 90-247-3626-9.

Wolfe, M.S. 2000. Crop strength through diversity. *Nature* 406, 681-682.

Yang, X.B. 2006. Framework development in plant disease risk assessment and its application. *European Journal of Plant Pathology* 115, 25-34.

Yarham, D. 1988. The contribution and value of cultural practices to control arable diseases. In: *Control of Plant Disease: Costs and Benefits* (Clifford BC, Lester E, eds.), 135-154.

Yildirim, A., Jones, S.S., Murray, T.D., Cox, T.S., Line, R.F. 1995. Resistance to stripe rust and eyespot diseases of wheat in *Triticum tauschii*. *Plant Disease* 79, 1230-1236.

Yuen, J., Hughes, G. 2002. Bayesian approaches of plant disease prediction. *Plant Pathology* 51, 407-412.

Zadoks, J., Schein, R.D. 1979. *Epidemiology and plant disease management*. Oxford University Press.

Zadoks, J.C. 1984. Analyzing cost effectiveness of EPIPRE. *EPPO Bulletin* 14, 401-407.

Zadoks, J.C. 1985. On the conceptual basis of crop loss assessment: The threshold theory. *Annual Review of Phytopathology* 23, 455-473.

Zhan, J., Mundt, C.C., McDonald, B.A. 2007. Sexual reproduction facilitates the adaption of parasites to antagonistic host environment: Evidence from empirical study in wheat-*Mycosphaerella graminicola* system. *Journal of Parasitology* 37, 861-870.

Zhang, X., Haley, S.D., Jin, Y. 2001. Inheritance of Septoria tritici blotch resistance in winter wheat. *Crop Science* 41, 323-326.

Zhang, X.Y., Loyce, C., Meynard, J.M., Monod, H. 2007. Modeling the effect of cultivar resisistance on yield losses of winter wheat in natural multiple disease conditions. *European Journal of Agronomy* 26, 384-393.

Zhao, F.J., Su, Y.H., Dunham, S.J., Rakszegi, M., Bedo, Z., McGrath, S.P., Shewry, P.R. 2009. Variation in mineral micronutrient concentrations in grain of wheat lines of diverse origin. *Journal of Cereal Science* 49, 290-295.

Acknowledgements

- To my supervisors Erland Liljeroth, Eva Johansson, Arnulf Merker and Jan-Eric Englund and my head of department Ylva Hillbur who gave me precious support.
- To my co-authors Jan-Eric Englund, Torbjörn Ewaldz and Håkan Rosenqvist, by your contributions the articles matured.
- To several people who have worked at the department doing disease assessments, handling data *etc.* for long periods Torbjörn Ewaldz, Per Nilsson and Lennart Pålsson and many more, have contributed to the results.
- To Björn Andersson and Torbjörn Leuchovius at the Field Research Unit (Fältforsk) who have made handling of data from field trials straightforward.
- To several people who have performed good field trials at the Agricultural Societies and to Skåneförsöken and its board, amongst others Per-Göran Andersson, Lars Håkansson, Arne Ljungars, Dave Servin and Hans Thorell.
- To Hans Andersson, Göran Gustafsson, Gareth Hughes, Lennart Johnsson, Arne Ljungars, Mary McAfee, Christer Nilsson, Lennart Pålsson, Gunnar Svensson and by anonymous reviewers for support and constructive criticism on the manuscripts.
- To Peder Waern who has taken all the first-class photos on plant diseases in this thesis.
- To Per Lundin, Jan Ö. Jönsson and Toma Magyarosi in discussions on host plant resistance and seed health.
- To several people at the Swedish Board of Agriculture and the Plant Protection Centres.
- To several people at Plant Protection companies for good cooperation.

- To Ulla Ekström and colleagues at the SLU library who have traced books, articles and been helpful in installing the template for the thesis.
- To Gary Nilsson and collegues at Repro.
- To The Swedish Farmers' Foundation for Agricultural Research, the Swedish Board of Agriculture and SLU who funded this research.

It is now 30 years since I began working at the experimental division of fungal and bacterial diseases at the Swedish University of Agricultural Sciences (SLU). Field trials were our method and plant diseases our object. These have been very interesting years and in spite of the 30-year period, time has been short. We started to look at fungicides in cereals in the late 1970s, first with sulphur against powdery mildew and then the systemic fungicides. In different ways, we have tried to optimise and minimise the input of fungicides. We have not been very successful in limiting the use of fungicides in favour of other control methods, but we have aimed at supervised control comprising other possible control measures. From these first years when I actually started my PhD work I am obliged to a number of people, at Ultuna Lennart Johnsson, Börje Olofsson, Hans Olvång and Vilhelm Umaerus, and at Alnarp Kjell Andersson, Stig Andersson, Hans Larsson, Bengt Leijerstam, Christer Nilsson and Guy Svedelius. At the interesting and enjoyable Nordic courses in plant pathology arranged by Vilhelm Umaerus, Viggo Smedegaard-Petersen, J. Helms Jørgensen, Leif Sundheim, Eeva Tapio and colleagues my interest deepened. These courses always had very good teachers including international experts such as A. Ellingboe, J. Dekker, Z. Király, G. Strobel and J. Zadoks.

Finally, I would like to thank all those not mentioned who have contributed over these 30 years.