Modelling effects of *Barley yellow dwarf virus* on growth and yield of oats

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Doctoral thesis
Swedish University of Agricultural Sciences
Uppsala 2006

Acta Universitatis Agriculturae Sueciae
Year: 2006: 16

ISSN 1652-6880
ISBN 91-576-7065-X

Tryck: SLU Service/Repro, Uppsala 2006

Abstract

Persson, T. 2006. *Modelling effects of Barley yellow dwarf virus on growth and yield of oats*. Doctoral thesis.
ISSN 1652-6880 ISBN 91-576-7065-X

Barley yellow dwarf (BYD) is a viral disease caused by a group of viruses that infect plant species within the family *Poaceae* and cause grain yield losses in cereal crops worldwide. The viruses causing the disease are divided into two groups, *Barley yellow dwarf virus* (BYDV) and *Cereal yellow dwarf virus* (CYDV), which are further divided into species and isolates. The viruses are spread from plant to plant only by grass feeding aphids.

A growth model describing oats (*Avena sativa*) infected with BYDV was formulated. Driving variables for the model are air temperature and solar radiation. The model consists of three sub-models, one describing plant phenology, the second development of green plant area and the third plant growth and biomass allocation between vegetative tissues and grains. Green plant area determining parameters, radiation use efficiency (RUE) and allocation parameters were calibrated against data from a greenhouse experiment. The model was modified, and RUE recalibrated to fit field data from an experiment with artificial BYDV infections in oats carried out at the Swedish University of Agricultural Sciences south of Uppsala (59°49'N/17°39'E), in 2002. The reductions in RUE calibrated against BYDV-infected plants were tested against grain yield data from another experiment carried out in 2003 at the same geographical site with the same oat cultivar and same virus isolate as in the experiment from 2002. To investigate the relative importance of air temperature and solar radiation in relation to other factors previously shown to influence the degree of grain yield reductions in B/CYDV-infected oats, grain yields from experiments in which cultivar, virus isolate and type of infection procedure differed from the Ultuna experiment 2002 were simulated.

The results of the model calibration show that green plant area determining parameters, RUE and allocation parameters are affected by a BYDV infection. In general, the test simulations of grain yield reductions differed considerably from those observed. Reasons for these differences and suggestions for model improvements are discussed in with help of experimental results on plant nitrogen changes in BYDV-infected plants.

Keywords: Avena sativa, BYDV, Barley yellow dwarf, plant growth, plant virus diseases, radiation use efficiency, RUE, simulation models

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Contents

Appendix

This thesis is based on the following papers, which will be referred to by their Roman numerals

I. Bisnieks, M., Persson, T., Eckersten, H. & Sigvald, R. 2005. The effects on yield and components of yield in oats infected with BYDV-PAV at different growth stages. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 112*, 521-528.

II. Persson, T. & Eckersten, H. 2003. Crude protein and lipid concentration in grains from oats infected with barley yellow dwarf virus. *The BCPC International Congress Crop Science and technology 2003* The British Crop Protection Council. Glasgow. UK.

III. Persson, T., Eckersten, H., Kvarnheden, A. & Yuen, J. Modelling influence of virus infection on leaf area and radiation use in oats under controlled climatic conditions. *Acta Agriculturae Scandinavica Section B-Soil and Plant Science. In press*

IV. Persson, T., Eckersten, H., Kvarnheden, A. & Yuen, J. Modelling virus effects on oat grain yield under field conditions. (*Submitted)*

Introduction

How has B/CYDV been studied in relation to its effect on crop yield?

Of the viruses infecting species within the family *Poaceae*, much attention has been paid to viruses causing the disease barley yellow dwarf (BYD). Viruses belonging to the group of positive-sense single-stranded RNA viruses (Comeau & Haber, 2004; D'Arcy, 1995) causing BYD are divided into two major groups, *Barley yellow dwarf virus* (BYDV) and *Cereal yellow dwarf virus* (CYDV). Barley yellow dwarf was first recognized and described in barley, oats and wheat in California by Oswald & Houston (1953) and named after its most conspicuous symptoms: dwarfish growth and leaf discolouration. It was concluded that BYD was caused by a virus and transmitted from plant to plant by aphids. Other types of transmission without involving an aphid vector did not seem to be possible (Oswald & Houston, 1953). Since that first report, the disease has been extensively studied. It has been reported from cereal crops, *e.g*. wheat, barley, oats, rye, maize and rice (Lister & Ranieri, 1995), as well as cultivated forage grasses and wild grasses on all continents (D'Arcy, 1995). Dwarfish growth and leaf discoloration in host plants have been further emphasized as traits of the disease, even though symptoms may vary considerably depending on plant species and cultivars infected (D'Arcy, 1995).

The effects of B/CYDV infection on plant function can be explained by anatomical and physiological changes. In its host, B/CYDV is limited to phloem tissue and occurs only in low concentrations compared with many other plant viruses (Jensen & D'Arcy, 1995). Accordingly, the most significant anatomical responses in B/CYDV-infected plants are cell death and degeneration of phloem tissue (Esau, 1957), effects which seem to be more severe after infections of young plants than of old (Panayotou, 1978). The phloem disruption leads to an inhibition of the transport of photo-assimilates from net assimilating tissues (source tissues) to tissues that are net importers (sink tissues) and thus an accumulation of photo-assimilates in source tissues. Photosynthesis also declines and chlorophyll content is reduced in B/CYDV-infected plants (Jensen & D'Arcy, 1995). In non-infected plants, an accumulation of carbohydrates leads to the degradation of chlorophyll and proteins involved in photosynthesis. Such an accumulation has been suggested to be an early step in senescence (Brouquisse *et al.*, 2001). Similar mechanisms might explain the decreased photosynthesis in BYDV-infected plants.

Plant viruses can alter nitrogen metabolism in their hosts, with effects on plant growth (Hull, 2002). As regards BYDV-effects, Orlob & Arny (1961) found a decrease in total nitrogen content and protein nitrogen in leaves of BYDV-infected plants of barley, whereas nitrogen content in roots of barley increased after an infection. During the vegetative growth phase of a plant, a major part of nitrogen in normally functioning leaves is linked to photosynthesis by constituting a component of enzymes involved in CO_2-assimilation (Lawlor, Lemaire & Gastal, 2001). Accordingly, a decline in both chlorophyll content and photosynthesis rate

has been found in BYDV-infected wheat (Jensen, 1972; Jensen & Van Sambeek, 1972). The effect of BYDV on grain protein concentration in infected plants can also be associated with the nitrogen metabolism. In grains from BYDV-infected barley (Edwards *et al.*, 2001), oats (Potter, 1980) and wheat (Fitzgerald & Stoner, 1967), nitrogen and protein concentrations have been reported to be higher than in grains from healthy plants. The higher nitrogen concentrations have been argued not to be a result of higher nitrogen contents in the infected plants, but rather a result of decreased starch filling in grains of infected plants (Jensen & D'Arcy, 1995).

Several environmental factors influence host responses to BYDV infection. After Oswald & Houston (1953) first found a relationship between growth stage of plant at infection and the degree of grain yield loss, these results were later confirmed by a great number of field studies with artificial B/CYDV infections of oats (Comeau, 1987; Doodson & Saunders, 1970; Endo & Brown, 1963; Gildow & Frank, 1988; Goulart, Ohm & Foster, 1989; Slykhuis *et al.*, 1959; Smith, 1967; Watson & Mulligan, 1960) and other cereal crops (Carrigan *et al.*, 1981; Comeau & St Pierre, 1975; Edwards *et al.*, 2001; Herbert *et al.*, 1999; Hoffman & Kolb, 1998; Huth, 1993) from different parts of the world. Studies dealing with B/CYDV-sensitivity among different host species indicate that oats often seem to be severely affected compared with wheat and barley (Lister & Ranieri, 1995; Pike, 1987). However, considerable differences in sensitivity between cultivars within the same cereal species have been found (Baltenberger, Ohm & Foster, 1987; Comeau & Dubuc, 1976; Doodson & Saunders, 1970; Goulart, Ohm & Foster, 1989), which can be referred to as differences in field tolerance (Burnett, Comeau & Qualset, 1995). There are also differences in effect on crop growth and yield between different virus species and isolates (Baltenberger, Ohm & Foster, 1987; Chay *et al.*, 1996; Gray, Smith & Sorrells, 1994). Temperature affects the rate of replication of various viruses and host species (Hull, 2002). Likewise, there are experiments showing temperature effects on BYDV-concentrations in plants (Forde, 1993). Secondary effects of temperature found after B/CYDV infections include *e.g.* a decreased winter hardiness in infected plants in winter cereals (Andrews & Paliwal, 1983; Paliwal & Andrews, 1979). Similarly, BYDV-infected plants are more sensitive to drought stress than healthy plants (Monneveux *et al.*, 1992), a consequence that has been speculated to be related to the negative impact of BYDV on root growth (Irwin & Thresh, 1990).

Selecting and breeding for tolerance (*i.e.* development of mild or negligible symptoms) against B/CYDV has in some countries been practised for many decades as a measure to limit yield losses (Burnett, Comeau & Qualset, 1995). Characters selected in such breeding programmes have included *e.g.* the ability of infected plants to stay green and the absence of effects on yield and yield components. These characters have been demonstrated in many cultivars of oats, barley, wheat and other cereal and grass species. However, many of these field tolerance cases have only been noted in studies at one or a few geographical sites, while no significant tolerance has been noted in other field experiments (Burnett, Comeau & Qualset, 1995). In barley, two genes: *Ryd2* (Rasmusson & Schaller, 1959; Sogaard & von Wettstein-Knowles, 1987) and *Ryd3* (Niks *et al.*, 2004) conferring field tolerance or resistance (reduced virus concentration) against

BYDV have been identified. However, the effects of BYDV have varied among cultivars containing the *Ryd2*-gene and field experiments (Burnett, Comeau & Qualset, 1995). The tolerance (Gill & Buchanon, 1972) and resistance (Skaria *et al*., 1985) in *Ryd2*-containing cultivars have been shown to be higher to BYDV-MAV and BYDV- PAV than to CYDV-RPV. Furthermore, in oats field tolerance has in some cases been associated with decreased virus concentrations (Skaria *et al.*, 1985).

More knowledge concerning taxonomic issues has been added to the original statements about the virus pathogen causing BYD. Rochow (1969) classified four main strains of BYDV, (PAV, MAV, RPV and RMV), based on the vector specificity (see next paragraph). Two new strains were later added to that classification, SGV (Gill, 1969; Rochow & Muller, 1971) and GPV (Zhang *et al*., 1983). Shepherd *et al*. (1976) recognized the virus group *Luteoviridae* in which the BYDV-strains were classified based on genetic properties. Today the classification based on vector specificity is still widely used and the main strains classified by Rochow, (1969) are defined as species (Lapierre *et al.*, 2004). Because of differences in nucleotide sequence and genome organisation among the BYD-causing viruses, which were originally classified as BYDVs, they have been divided into two distinct groups: *Barley yellow dwarf virus* (BYDV) and *Cereal yellow dwarf virus* (CYDV). PAV and MAV are still classified as BYDVs, whereas RPV and GPV are classified as CYDVs. The classification of the SGV and RMV species is not totally clear (Lapierre *et al.,* 2004). There are also differences in the nucleotide sequences among isolates within the same virus species (Bisnieks *et al.*, 2004; Chay *et al.,* 1996).

Extensive studies carried out on B/CYDV have further confirmed the persistent and obligate aphid transmission of the disease indicated by Oswald and Houston (1953). At least 28 different species within the family *Aphididae* have been shown to be effective vectors of one or several BYDV or CYDV-species (Harrington, 2002). Transmission tests on a few of the most notable vector species show that *Rhopalosiphum padi* is an efficient vector of BYDV-PAV and CYDV-RPV. *Sitobion avenae* (previously named *Macrosiphum avenae*) is an efficient vector of BYDV-MAV and relatively efficient vector of BYDV-PAV. *Rhopalosiphum maidis* is an efficient vector of RMV and *Schizaphis graminum* efficiently transmits SGV (Power & Gray, 1995; Rochow, 1969). These differences in transmission efficiency among vectors are, at least to some extent, reflected in a co-occurrence of different vector and virus species. In winter barley in western France, the occurrence and spread of BYDV-PAV have been shown to be linked to the population dynamics of the aphid species *R. padi, S. avenae* and *Metopolophium dirhodum* and the occurrence and spread of BYDV-MAV to the population dynamics of *S. avenae* and *M. dirhodum*, whereas no clear correlation was found between the dynamics of CYDV-RPV and *R. padi*, its main vector (Leclercq-Le Quillec *et al.*, 2000).

A BYDV infection in a field starts with colonisation by winged aphids, which often originate from very distant fields (Kendall, Brain & Chinn, 1992). Later, wingless aphids crawling on the ground from plant to plant spread the infection short distances within the infected field (Chaussalet *et al.,* 2000). Kendall, Brain &

Chinn (1992) showed in a simulation model validated under field conditions that long- and short-distance spread of BYDV to new plants is dependent on the number of vectors, the frequency of viruliferous vectors and their acquisition rate of BYDV. Another field study based model describes how the probability of a plant being infected is correlated with the infection status of its nearest neighbours (Chaussalet *et al.*, 2000). Theoretical model simulations indicate that an increased patchiness in the spatial distribution of BYDV would lead to a decrease in the rate of the virus spread (McElhany, Real & Power, 1995). There are also theoretical indications that the preference of vectors for infected or uninfected plants is of importance to the disease spread. In an environment with a high BYDV incidence, vectors preferring healthy plants favour the disease spread, whereas vectors preferring diseased plants favour the spread in an environment with low disease incidence (McElhany, Real & Power, 1995).

Control measures against BYDV have largely focused on preventing spread of the disease to new plants and minimizing the negative effects in infected plants. The control of vectors has therefore constituted a major control strategy against BYDV (Irwin & Thresh, 1990; Plumb & Johnstone, 1995). Rapid seed establishment and a closed canopy cause micro-environments that are less attractive to aphids species acting as BYDV-vectors (Plumb & Johnstone, 1995). Such effects can be achieved by good sowing techniques and early sowing. In spring-sown cereals, the latter measure has been found to be an efficient way of avoiding or limiting BYDV-induced yield reductions (Jenkyn & Plumb, 1983). In many regions, the earlier the sowing of spring cereals, the older and less attractive the plants when aphid populations normally peak (Plumb & Johnstone, 1995). Another effect associated with sowing time is, as mentioned above, that BYDV causes less grain yield reduction after infection at late growth stages than after infections at earlier growth stages. However, early sowing of winter cereals has in many regions been found to be negative, since it means that plants are often in a vulnerable growth stage at the autumn peak in the population of many vector species (Lowe, 1967; Plumb, 1992; Plumb & Johnstone, 1995).

Insecticide treatments of vectors can often be an efficient measure to limit BYDV-caused yield losses in cereal crops, especially when high yields are expected. Such treatments are particularly efficient if they are carried out to prevent secondary spread by wingless aphids from initial infection foci (McGrath & Bale, 1990; Plumb & Johnstone, 1995). Insecticides with persistent effects have proven more efficient against negative effects of BYDV than less persistent insecticides (McGrath & Bale, 1990). Seed dipping with insecticides has also been found to be an efficient way to reduce BYDV-spread (Gourmet *et al.*, 1996; Gray *et al.*, 1996).

Why should growth modelling be applied to study BYDV?

Various mechanistic growth models aimed at simulating the dynamics of cereal growth and yield have been published over recent decades (Amir & Sinclair, 1991a; Eckersten & Jansson, 1991; Jamieson *et al.*, 1998b; McCown *et al.*, 1996; Porter, Jamieson & Wilson, 1993; Ritchie & Otter, 1985; Weir *et al.*, 1984). Such models can be regarded as formalised collections of testable hypotheses about how

environmental factors affect plant growth processes (Jamieson *et al.*, 1998a). Models aimed at simulating cereal crops have been created to explain complex biological systems by simplifying them in mathematical equations. Sub-systems in crops are modelled and linked together to mechanistically describe the function of the system as a whole. The greatest emphasis in cereal crop modelling has been placed on simulating wheat, rice and maize. Thanks to similarities in the physiology of species within the family *Poaceae* and because crop simulation models have rather simple structures, it has been suggested that such models could be applied to also simulate other cereal species such as barley and oats (Jamieson *et al.,* 1998b).

There are several existing process based models aimed at simulating epidemics and yield losses caused by various plant pathogens in cultivated crops. In contrast to the plant growth models outlined in the paragraph above, such models are often centred on environmental factors that regulate the appearance and growth of the pathogen instead of the host plant. Factors that regulate disease outbreaks in these types of simulation models are, for example, temperature, soil moisture and occurrence and behaviour of disease vectors (Agrios, 1997). The simulation models of BYDV-spread outlined above (Chaussalet *et al.*, 2000; Kendall, Brain & Chinn, 1992; Leclercq-Le Quillec *et al.*, 2000; McElhany, Real & Power, 1995) are examples of models where vector occurrence and behaviour control the disease outbreak. However, this type of model does not take into account the response of an infected plant to different environments. Depending on the extent to which such effects contribute to yield losses in infected plants, the predictability of disease simulation models could be increased by taking these effects into account. As mentioned above, grain yield reductions in B/CYDV-infected oats vary considerably among experiments. Environmental factors are suggested to be of importance for the magnitude of grain yield reductions in cereals. In contrast to the vector influence on disease outbreak, there are no efficient tools to predict the impact of environmental factors on yield losses in B/CYDV-infected cereals.

The idea of coupling diseases to plant growth models have previously been emphasised (Boote *et al.*, 1983; Pinnschmidt, Teng & Yuen, 1990; Rouse, 1988). The relevance of models that simulate pathogen impact, but that are still centred on the attacked or infected crop, should be highest when they describe the effects of pathogens that alter plant growth without causing plant death or total yield reductions. Plant growth models simulating diseased plants do not give much extra information in the understanding of pathogens that are completely devastating to their host plants. There are a few examples of linking epidemiology models of specific pathogens to weather-driven simulation models of cereals. Pathogen impact has been coupled to photosynthesising leaf area when integrating a fungal disease epidemiology model (BLASTSIM) into a crop growth model (CERES-Rice) (Luo *et al.*, 1997). There are also examples of coupled models describing the impact of the fungi *Stagonospora nodorum* (Djurle & Yuen, 1991) and *Erysiphe graminis* (Rabbinge, Jorritsma & Schans, 1985) on winter wheat growth.

Unlike many other plant pathogens, viruses do not produce toxins or other pathogenic substances (Agrios, 1997). The presence of a viral pathogen in a plant does not automatically lead to any disease symptoms. In general, disease

symptoms caused by plant viruses are indirectly induced changes in the plant metabolism (Agrios, 1997). Since the effects of plant viruses on their hosts are rarely lethal (Agrios, 1997), it would make sense to study host responses of plant viruses in plant growth simulation models. Virus effects on photosynthesis have accordingly been simulated in a weather-driven growth model describing the interaction between sugar beet and *Beet yellows virus* (BYV) (van der Werf, 1988). The indications that B/CYDV effects on plant growth and magnitude of yield losses are weather-dependent (Irwin & Thresh, 1990) make it meaningful to also study the effects of this viral pathogen in a weather-driven plant growth model.

A model that aims at predicting yield losses in B/CYDV-infected oats should simulate plant growth and development so that virus effects could be represented in sufficient detail to predict the impact of the disease on growth and grain yield. Patterns in plant growth that are changed by an infection so that they affect grain yield should preferably be simulated. Naturally, the more detailed the disease impacts on the infected plant simulated, the higher the understanding of the environment-plant-virus interactions. Simulations of molecular interactions or interactions on even smaller levels would give the best representation of the plant-virus-environment system and theoretically the highest predictability. However, such studies would be too complex to handle. There is limited knowledge about interactions on these levels and studies aimed at investigating them would hardly be testable. Therefore, disease-induced changes in plant physiology or anatomy could rather be simplified and represented by effects on a higher level, as long as those simplifications do not affect the simulated grain yield losses. Disease impact on plant physiology that does not directly affect grain yield could be represented by impact on a higher scale.

The result of van der Werf's (1988) simulation of virus infected sugar beet was an almost complete inhibition of photosynthesis in discoloured leaves. This inhibition was the most important factor in the reduction in plant growth and in root and sugar yield, whereas healthy leaves of the infected plants photosynthesised at normal rates. Similarly, the simulation of BYDV impact on crop growth could be based on how physiological and anatomical effects of the pathogen can be scaled up and represented on a crop stand level. Symptoms of BYDV-infected plants give hints about where in the model structure the disease impact should occur so that the diseased crop would be represented in a sufficiently realistic way to predict grain yield reductions.

Growth models aimed at simulating healthy cereal crops consist of testable hypotheses about the impact of environmental factors on growth and yield, formalised in mathematical equations. The structure among models varies considerably depending on the objective of the model (Jamieson *et al.*, 1998a). Development of basic model structures describing the impact of weather on crop growth structures has been initiated to mechanistically answer questions about how solar radiation and air temperature regulate variation in grain yield of wheat between sites and growth seasons (Porter, 1985). Such basic structures would also be vital to a model describing the interactions between weather conditions and virus impact on crop growth and yield. However, models that only take into

consideration air temperature and solar radiation as factors which affect plant growth (Amir & Sinclair, 1991a; Weir *et al.*, 1984) presuppose that the state of other environmental factors that may limit crop growth is optimal for crop growth. In addition, such models cannot simulate changes in quality parameters (*e.g.* protein concentration) in the harvested yield due to changes in environmental conditions that do not affect the quantitative yield. Further model development has included simulations of nitrogen uptake and transport in the crop in order to predict not only yield quantity but also, for example, plant nitrogen content (Jamieson & Semenov, 2000; Jamieson *et al.*, 1998b; Jamieson, Stone & Semenov, 2001; Porter, 1993; Sinclair & Amir, 1992). Similar to nitrogen effects, modelling effects of water limitation to crop growth enlarges the potential use of simulation models to geographical regions that are often exposed to low precipitation rates and to model differences in water availability between geographical sites and growth seasons (Amir & Sinclair, 1991b; Jamieson *et al.*, 1998b; Porter, 1993). From the perspective of simulating plant growth, diseases, similarly to water and nutrient availability, have been suggested as important environmental factors that could be taken into account in cereal crop models (Ritchie & Otter, 1985).

Attempts have also been made to model other environmental effects, notably farming practices (*e.g.* sowing time), that may indirectly affect the weather conditions under which the crop is grown (Porter, 1985). The objective of simulating indirect effects of farming practices in cereal simulation models has been further stressed in model development aimed at creating useful tools for decision making in practical farming (Ritchie & Otter, 1985). Simulating diseased plants too would enlarge the potential for implementing growth models into different situations in practical farming.

What model characteristics would be useful when simulating BYDV impact?

Despite diverging objectives, there are a few processes of central importance and these are formulated in most cereal growth models. Stages in plant phenology are set as a function of temperature sums and in a few cases also day length. These stages regulate changes in other sub-models (Amir & Sinclair, 1991a; Jamieson *et al.*, 1998b; Ritchie & Otter, 1985; Weir *et al.*, 1984). The size of green plant area in a crop has often been expressed as the green plant area over a specified ground area, the leaf area index (LAI) or more correctly the green plant area index (GAI). This concept is another of the basic principles incorporated into cereal crop simulation models. However, the way the development of GAI over time is calculated differs among models. GAI has been described in plant growth models as a function of the number of leaves per plant which, in turn, is related to the air temperature and day length (Amir & Sinclair, 1991a; Porter, 1984). A second way is to let GAI be directly linked to the environmental driving forces of the model (Jamieson *et al.*, 1998b). In a model of BYDV impact, visible symptoms of the pathogen indicate that the disease factor could be modelled by changing parameters that regulate GAI.

Plant or crop growth, defined as increase of biomass over time, can be described as a function of intercepted radiation. The concept radiation use efficiency (RUE), which is the factor by which biomass is produced per amount of intercepted solar radiation (physiologically based on photosynthesis reactions) was first introduced during the 1950s (Sinclair & Muchow, 1999). Later, RUE has been included in cereal growth models as a driving parameter (Amir & Sinclair, 1991a; Jamieson *et al.*, 1998b; Ritchie & Otter, 1985; Weir *et al.*, 1984). The radiation used for plant biomass production is intercepted by the green photosynthesising plant area. As mentioned above, nitrogen concentration in above-ground vegetative plant tissues is linked to the photosynthetic capacity since a major part of nitrogen in these tissues is bound in proteins involved in photosynthesis (Lawlor, Lemaire & Gastal, 2001). In turn, the concentration of nitrogen per green plant area is controlled by light intensity. Nitrogen effects on plant growth have therefore been suggested to be modelled so that nitrogen demand is linked to green plant area expansion in order to keep the nitrogen concentration per leaf area constant (Grindlay, 1997). A simpler approach included in cereal models has been to regulate plant nitrogen demand and concentration with plant phenology (Jamieson *et al.*, 1998b; Porter, 1993; Sinclair & Amir, 1992). A later method of modelling plant nitrogen divides the nitrogen demand into different above-ground plant organs (Jamieson & Semenov, 2000). This method is more similar to that suggested by Grindlay (1997) since nitrogen demand is regulated by green area and stem demand. Likewise, shortages of nitrogen first affect GAI-expansion and later RUE (Jamieson & Semenov, 2000). BYDV-induced responses in photosynthesis as reported from practical experiments (Jensen, 1972; Jensen & Van Sambeek, 1972), and possibly linked to alterations in plant nitrogen status, could be modelled in a similar way by letting the pathogen affect not only GAI-expansion as indicated by visible symptoms, but also by changing RUE.

Objective

The main objective of this thesis was to formulate and test a mechanistic weather-driven model describing growth and development of oats infected with *Barley yellow dwarf virus* (BYDV) under controlled conditions and field conditions. A second objective was to assess grain yield losses and changes in the composition of grains from BYDV-infected oats grown under Swedish weather conditions. A third objective was to measure plant nitrogen concentrations in BYDV-infected oats and compare nitrogen concentrations with grain yield reductions.

Materials and methods

The work consists of model formulation, model calibration to greenhouse and field conditions and tests of the model. Model input and calibration data were to a large extent obtained from practical experiments carried out in connection with the modelling work.

Experimental work

Greenhouse experiment

The model calibration was performed against data from a controlled greenhouse experiment in which oats (cv. Stork) were grown (see Paper III for details). The experiment was set up with a complete randomised design. Four treatments were included: BYDV infection of oat plants at growth stage (GS) 12 (Zadoks, Chang & Konzak, 1974) with a high fertilisation regime, infection at GS 12 with a low fertilisation regime, uninfected plants with a high fertilisation regime and uninfected plants with a low fertilisation regime. Radiation above and within the canopy and air temperature were measured during the whole growth period for use as input in the model calibration. The BYDV infections were carried out 16 days after sowing by transferring approximately five aphids to each experimental plant in the treatments to be infected. A few days later, the aphids were killed with an insecticide. At four times during the growth period, randomly chosen plants were harvested. The model was calibrated against data on above-ground plant dry weight and green plant area from these four measurements. Plant nitrogen content was also analysed at the time of measurement.

Field experiments

The data for the model recalibration into field conditions were taken from an experiment carried out south of Uppsala, Sweden, in 2002. In this experiment there were five treatments, infections in growth stages (GS) 11, 13, 31 and 39 (Zadoks, Chang & Konzak, 1974) and uninfected control plants. After harvest at full plant maturity, above-ground vegetative and grain yields were measured, and protein, lipid, calcium, phosphorus, potassium and magnesium concentrations in the grains were analysed. (For a full description of the experiment see Papers I and II). The oat cultivar (Stork) grown was the same as in the greenhouse experiment. In an experiment from 2003 with the same oat cultivar as in the experiment from the previous year, the same virus isolate as was used in 2002 was again included, together with treatments with three other BYDV-isolates.

Modelling work

A model describing growth and development of BYDV-infected oats and driven by air temperature and solar radiation was formulated and constructed in Powersim Studio 2003 ® (Powersim AS, Bergen, Norway). Below, the main principles formulated and the calibration and tests of the model are briefly summarized. (For a full description of the work, see Papers III and IV.) The basic model structures were adapted from previously published models describing wheat (Amir & Sinclair, 1991a; Brooking, Jamieson & Porter, 1995; Jamieson *et al.*, 1998b). Parameters to fit growth and development of spring oats were set according to observations in the greenhouse and field experiments and according to literature data (Peltonen-Sainio, Forsman & Poutala, 1997) considered

applicable. Driving variables in the model are solar radiation, air temperature and day length. The model is divided into three sub-models, one describing plant phenology, a second development of the green plant area and the third plant growth and allocation between vegetative and grain biomass (Fig. 1).

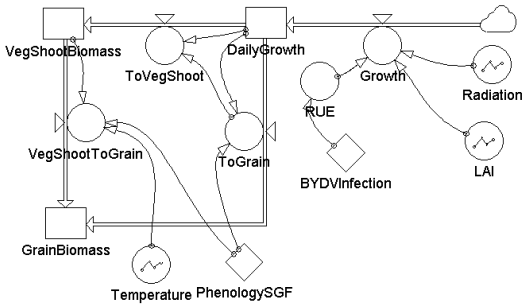

Fig. 1. a) Growth and allocation sub-model (From Persson *et al.* (2006)

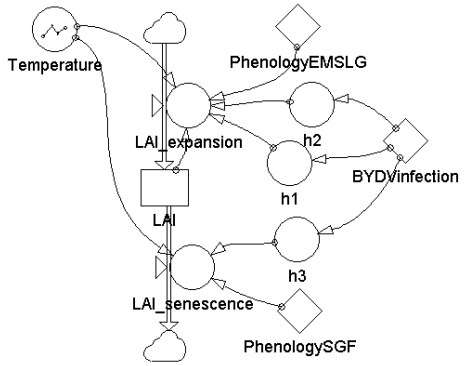

Fig. 1 b) Green plant area sub-model. (From Persson *et al.* (2006)

Phenological stages are determined by switches based on sums of daily air mean temperature and, in field applications, also day length. Phenological stages initiate changes in the two other sub-models; a connection describing how green plant area development and plant growth are responding to air temperature and day length. The green plant area development is driven directly by air temperature, and indirectly by phenological stages. The BYDV infection is imposed on the model by affecting the development of green plant area. The relative change in the green plant area development factor is:

$$fh_{BYDV}=h_{BYDV}/h \qquad (1)$$

where h is the factor regulating the green plant area expansion and decrease. The green plant area is also linked to growth by determining the intercepted solar radiation. Intercepted solar radiation is conditioned by stand density and is either calculated as the mean of radiation within the canopy multiplied by GAI, or by Lambert-Beer's law (Monteith & Unsworth, 1990). The base of the linkage between green plant area and growth is that assimilated biomass is calculated by

multiplying the intercepted solar radiation by a factor called radiation use efficiency (RUE). Similar to the BYDV-effect on parameters determining green plant area, RUE is also affected. The relative change in RUE in infected plants is:

$$f_{\varepsilon BYDV} = \varepsilon_{BYDV} / \varepsilon \quad (2)$$

where ε_{BYDV} is RUE of infected plants and ε is RUE of uninfected plants. All assimilated biomass is allocated to the vegetative pool before grain filling, and in uninfected plants, after the beginning of grain filling, all assimilated biomass is allocated to the grain pool. In BYDV-infected plants, a fraction of biomass assimilated during grain filling is allocated to the vegetative pool. The relative change in grain growth of infected plants is:

$$fl_{gfBYDV} = (\Delta W_{grain}/\Delta t)_{infected} / (\Delta W_{grain}/\Delta t)_{uninfected} \quad (3)$$

where $\Delta W_{grain}/\Delta t$ is the growth of grain biomass in infected and uninfected plants, respectively. During grain filling, there is also a translocation of biomass from the vegetative biomass pool to the grain pool. This translocation is not affected by the BYDV infection.

Model calibration to greenhouse experiment

The calibration of parameters in the model was carried out against observations in the greenhouse experiment or literature references (Jamieson *et al.*, 1998b; Sonego *et al.*, 2000). (See Paper III for details.) First, switches for the phenological stages were calibrated. These switches were based on sums of daily mean air temperature. Secondly, the parameters determining expansion and decrease of the green plant area were calibrated. The calibration of the green plant area sub-model was first performed against uninfected plants. Subsequently, the BYDV-factor (fh_{BYDV}; eq. 1) was calibrated against observations in infected plants. Radiation use efficiency (RUE) was calibrated in a similar way. First, RUE was calibrated against the above-ground biomass of healthy plants. Next, the deviation between observed and simulated above-ground dry matter in BYDV-infected plants was simulated by changing RUE in the infected plants ($f_{\varepsilon BYDV}$; eq. 2). The parameters were calibrated separately for the different phenological stages. In addition, for the infected plants, the parameter that regulates the allocation of biomass between the vegetative and the grain pool was also calibrated against the ratio between grain and vegetative biomass (harvest index) at plant maturation.

Model applications to field conditions

The model was also applied to field conditions. This application consisted of a few modifications of the model structure and a recalibration against data from an experiment with controlled BYDV infections of oats. The recalibrated model was also tested against independent data from experiments where oats were artificially infected with BYDV or CYDV and designed similarly to the calibration experiment. A sub-module describing the day length dependence in the phenology sub-model was added (see Paper IV) to allow experiments from different latitudes to be simulated. Another major change from the greenhouse calibration was that RUE was constant over the whole growth season.

The recalibration of the model was performed against an experiment where oats (cv. Stork) were infected with an isolate of BYDV-PAV at the Swedish University of Agricultural Sciences (59°49'N/17°39'E), Ultuna, south of Uppsala in 2002. (See Paper I for experimental details and Paper IV for calibration details.) The BYDV-effects on RUE were tested against grain yield reductions in oats (cv. Stork) grown in an experiment in 2003 at the same site as the experiment from 2002. The virus isolate used for infection was identical to that used in 2002. The test was carried out so that the model was first recalibrated against uninfected control plants and subsequently applied to infected plants using the parameterisation of 2002. Temperature and radiation input data to the model calibration and the model test were obtained from Ultuna climate station, approximately 500 metres southwest of the experimental site.

The objective of the study was also to test the model predictability under field conditions when virus isolate, oat cultivar, growth stage at infection and length of infection period varied. In single experiments, all these factors have been related to the magnitude of grain yield reduction in BYDV- or CYDV-infected oats. This can be regarded as an attempt to assess the predictability of the model against at least a part of the variation of the oats-B/CYDV-system as it occurs worldwide. The model was not designed to account for these variations, but the model application is expected to highlight the importance of cultivar, virus type and length of infection period when weather impact is simulated. The model was first tested against isolates included in the infection scheme of the 2003 experiment that differed from that used in the calibration experiment. In order to also obtain a variation in the other factors stated above, a screening of publications treating spring oats artificially infected with BYDV or CYDV was carried out (Table 1). The importance of these factors to the grain yield reductions in B/CYDV-infected oats was tested with two methods, one statistical and one model simulation. To allow data from experiments with different plot sizes to be compared, grain yield reduction was defined as the ratio of grain yield of the infected plants to the yield of uninfected control plants (relative grain yield reduction) in the respective experiment. The importance of cultivar, growth stage at infection, virus species and length of infection period to the relative grain yield reduction was tested in general linear model (GLM) analyses performed in SAS v. 8.01 (SAS Institute Inc, Cary, NC, USA). Two different GLM-analyses were carried out. In the first analysis, all factors mentioned above, except virus species, were analysed by including all observations from all experiments. The virus species was excluded since information about that factor was missing in the oldest references. The second analysis was carried out including only the treatments where the virus species used for infection could be defined as BYDV-PAV, CYDV-RPV or a mixture of those two species. Missing information about *e.g.* exact geographical site and time of experiment in many of the reference experiments screened made it impossible to simulate grain yields from all the experiments in Table 1. Data from the experiments published by Baltenberger, Ohm & Foster (1987) and Gildow and Frank (1988) were presented in a way that made it possible to simulate relative grain yield reductions in infected treatments after recalibrations to the respective uninfected control treatments.

Table 1. *References included in the statistical analysis*

Original reference	Experi-mental site	Year	Oat cultivars	B/CYDV species	Plant growth stage (GS) at infection[1]	Infection period (days)
(Slykhuis *et al.*, 1959)	Ottawa, Canada	1958	Clintland, Garry	Unknown	13-14	7
(Watson & Mulligan, 1960)	Rothamsted, UK	1956, 1957	Blenda, Milford	Unknown	23[2], 39[2]	7
(Endo & Brown, 1963)	Urbana, IL, USA	1958	Clintland, Newton, Albion, Saia	Unknown	13-14, 31, 39, 45	3
(Smith, 1967)	New Zealand	1961	Clintland	RPV	11, 39	3
(Doodson & Saunders, 1970)	Cambridge, UK	1965, 1966, 1968, 1969	Blenda, Condor, Manod, Mapua	Unknown	11, 23, 31, 39	3
(Jedlinski, 1972)	Urbana, IL, USA	1971	Clintland, Newton, Albion, Saia	PAV	23	3
(Baltenberger, Ohm & Foster, 1987)	West Lafayette, IN, USA	1983	Ogle, Clintland, Porter, Acc1575	PAV, RPV, PAV and RPV	11	5
(Gildow & Frank, 1988)	Centre County, PA, USA	1985, 1986	Noble	PAV	23[2], 39[2]	7
(Goulart, Ohm & Foster, 1989)	West Lafayette, IN, USA	1987-1988	Ogle, 7869D1-5-3-4, Noble, Putnam 61, Clintland	PAV	12-13, 13-14, 14-15, 39	14
(Bauske, Bissonnette & Hewings, 1997)	Urbana, IL, USA	1991, 1992	Ogle, Noble	PAV	23	7
(Bisnieks *et al.*, 2005)	Ultuna, Sweden	2002	Stork	PAV	11, 13, 31, 39	7
See description below	Ultuna, Sweden	2003	Stork	PAV	11, 31	7

[1] GS according to Zadoks, Chang & Konzak,. (1974)
[2] Estimated, given as days after sowing in the original publication

Results

Experimental results

Green plant area, above-ground biomass and grain yields

There were statistically significantly smaller green plant areas in the infected plants than in the uninfected plants in the two nitrogen treatments in the greenhouse experiment. This effect decreased as the plants grew older (Paper III). Both in the greenhouse experiment and the field experiment from 2002, total above-ground biomass and grain yields were in most cases lower in the infected plants than in the uninfected plants. One exception was in the field experiment from 2002, where the above-ground biomass in plants infected at GS 39 was higher than in uninfected plants at maturation (Paper I). In the highly fertilised plants in the greenhouse, total above-ground biomass was also higher in infected plants at maturation than in uninfected plants. A tendency observed in the field experiment was that the earlier the infection, the greater the above-ground biomass and grain yield reductions (Paper I).

Nitrogen, lipid and mineral concentrations

In the greenhouse experiment, both in BYDV-infected and uninfected plants, the nitrogen concentration expressed per unit dry weight in above-ground tissues decreased as the plants grew older. However, the nitrogen concentration expressed per unit of green plant area, in general, increased over time. Another overall tendency was a relative increase in nitrogen concentration (per m^2 green plant area) in the infected plants, compared to the uninfected plants, as the plants grew older (Paper III). In the field experiment, crude protein concentration was significantly higher in grains from the infected plants than in grain from the uninfected plants. However, the concentration did not differ significantly among plants infected at different growth stages. Grain lipid concentration was significantly lower in plants infected at GS 11, 13 and 31 compared with uninfected plants. Concentrations of phosphorus were significantly higher in grains from plants infected in GS 11, 13 and 31 than in grains from uninfected plants and potassium was higher in grains from plants infected in GS 11, 13 and 39 than in grains from uninfected plants. There was no statistically significant difference in calcium and magnesium concentration in grains among treatments (Paper II).

Model calibration

The model calibration against greenhouse observations showed that parameters determining the development of green plant area are affected by a BYDV infection (Table 2). Effects of BYDV on RUE were calibrated as shown in Table 2 to minimize the discrepancy between simulated and observed biomass. The deviation between observations and simulations that nevertheless occurred can be ascribed to differences between measurements and switches in the phenology sub-model. The fraction determining allocation to vegetative biomass was higher in infected

than in uninfected plants in both fertilisation regimes. In the adaptation of the model to the field experiment of 2002, the mean of green area in infected plants over the growth period varied from 62% (in GS 11 treatment) to 102% (in the GS 39 infection) of that in the uninfected plants. The reduction in RUE varied among treatments. The later the growth stage at infection, the higher the RUE and the smaller the reduction (Table 2). Similarly, the relative value of the allocation coefficient determining the biomass allocation to grains was higher in later infections (Paper IV).

Table 2. *Calibrated changes in green plant area parameters and RUE in BYDV-infected plants*

	Greenhouse calibration (Paper III)		**Field calibration (Paper IV)**			
BYDV-effect	**Low fertilisation plants**	**High fertilisation plants**	**GS 11**	**GS 13**	**GS 31**	**GS 39**
Rel. change in h_1	0.626	0.581	0.625	0.625	0.625	0.625
Rel. change in h_2	1.09	0.943	1.09	1.09	1.09	1.09
Rel. change in h_3	0.872	0.799	0.717	0.789	0.926	1.04
Rel RUE (total growth period)	-	-	0.607	0.747	0.886	1.09
Rel RUE (before EMSLG[1])	0.589	0.773	-	-	-	-
Rel RUE (between EMSLG and anthesis)	1.77	1.74	-	-	-	-
Rel RUE (during anthesis)	0.194	0.138	-	-	-	-
Rel RUE (during grain filling)	1.19	2.76	-	-	-	-

[1] EMSLG= End of main stem leaf growth

Model tests and statistical analyses

After the recalibration of RUE of uninfected plants against the controls in the 2003 experiment, the application of relative RUE reductions from the experiment at Ultuna in 2002 resulted in 26% higher relative grain yield than observed in the treatment with the same virus isolate as 2002. Introducing new virus isolates, oat cultivars and geographical locations of the experimental site that differed from the calibration data set resulted in increased deviations between simulated and observed relative grain yields in most simulations. The general predictability evaluated in a regression analysis of all simulated relative grain yield reductions and respective observations showed a low R^2-value (0.16) (Fig. 2) (Paper IV). Likewise, the statistical analyses including a broader data set than in the model

tests showed that growth stage at infection, virus strain, oat cultivar and length of infection period are all of importance for the reduction of relative grain yield in B/CYDV-infected plants (Table 3).

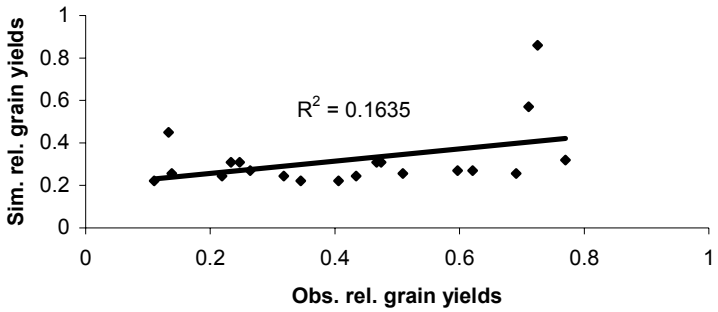

Fig. 2. Simulated versus observed relative grain yield including all test data from Ultuna 2003 and from Baltenberger, Ohm & Foster (1987) and Gildow & Frank (1988).

Table 3. *Results of GLM-analyses*

First analysis			
No. of observations included	*91*		
Type of analysis	*GLM type III Sum of Squares*		
Factor tested	No. of treatments	F-value	Pr-value
Cultivar	17	2.96	0.0011
Growth stage at infection	8	9.43	<0.0001
Infection period	5	5.24	0.0010
Second analysis			
No. of observations included	*55*		
Type of analysis	*GLM type III Sum of Squares*		
Factor tested	No. of treatments	F-value	Pr-value
Cultivar	11	3.60	0.0028
Growth stage at infection	7	3.90	0.0049
Infection period	5	2.63	0.0527
Virus species	3	4.96	0.0133

Discussion

Model calibration and experimental results

The first findings of the model calibration against BYDV-infected plants was a decrease in the parameters regulating the rapid expansion of green plant area of young plants (Rel. h_1; Table 2). This stunting effect could be a consequence of the phloem degeneration that BYDV causes in infected plants (Esau, 1957; Panayotou, 1978), which limits the ability of infected plants to transport photo-assimilates to newly developed tissues such as expanding leaves (Jensen & D'Arcy, 1995). A slight recovery in green plant area expansion in infected plants during the slower second expansion phase suggests that oats have the ability to recover some time after infection. Skaria, Lister and Foster (1984) detected a decline in the concentration of BYDV-PAV-isolates in barley, oats and wheat after a peak in concentration about 12 days after the infection. If anatomical changes in phloem tissue are linked to virus concentration, the recovery in green area expansion might be a consequence of a decrease in virus concentration.

There are several possible reasons for the effects of BYDV infection on RUE. The greenhouse calibration showed that RUE, similarly to the green plant area expansion parameter, initially decreased after the infection, but was relatively higher than in uninfected plants during later phenological stages. This pattern of BYDV effects on RUE parallels a relative increase in plant nitrogen concentration per green plant area. In healthy plants, the rate of photosynthesis is positively correlated to plant nitrogen concentration expressed per unit green plant area (Grindlay, 1997). The increase in RUE in BYDV-infected plants might therefore be a result of an increased nitrogen concentration. In healthy plants, the relationship between nitrogen concentration, rate of photosynthesis and growth is based on the fact that a major part of the nitrogen in vegetative tissues is located in proteins involved in photosynthesis (Lawlor, Lemaire & Gastal, 2001). Experimental results regarding nitrogen and protein in BYDV-infected plants are somewhat contradictory. Orlob and Arny (1961) found decreases in total protein nitrogen in leaves of BYDV-infected barley, whereas Jensen (1969) reported unchanged content of soluble nitrogen in leaves of BYDV-infected barley. More experiments on protein function in BYDV-infected plants subject to controlled nitrogen supply would help explain mechanisms regulating effects of BYDV on plant growth.

There were problems in measuring radiation conditions within the greenhouse. The radiation conditions may have varied between infected and uninfected plants, *e.g.* due to differences in self-shading. To evaluate whether the effects of BYDV could be ascribed to the problems in radiation measurements, two contrasting methods were used to calculate intercepted radiation. However, the sensitivity of BYDV effects on RUE to the difference in intercepted radiation between the two methods was low (Paper III). Nonetheless, in healthy plants, the nitrogen concentration optimal for photosynthesis is linked to light intensity (Hirose & Werger, 1987). Consequently, increased light intensities in infected plants might have triggered nitrogen uptake. Smaller green area in plants and decreased self-

shading could thus have been the reason for the relatively higher nitrogen content per green plant area in infected plants. The contrasting RUE decrease in infected plants between the greenhouse experiment and the field (Paper IV) could be due to dissimilar effects of BYDV on light conditions. The plant density was higher by far in the field experiment and differences in densities between infected and uninfected plants were probably lower. Accordingly, light effects on plant nitrogen and photosynthesis in infected plants would have been less pronounced, which would entail a decreased possibility to compensate for decreased green plant area by a higher rate of photosynthesis. Another possible explanation for the differences in BYDV effects on RUE between the greenhouse and the field is that RUE was incorrectly simulated. Possible errors in the estimations of green plant area in the field experiment, due to the lack of observations, would have been transferred to RUE. The impact of growth stage at infection was taken into account in the calibration of RUE in the field application. In contrast, the impact of BYDV on green plant area determining factors was assumed to be identical in all infection growth stages simulated. However, effects on plant height of BYDV infections at different growth stages in this study (Paper I) and in other studies (Endo & Brown, 1963; Goulart, Ohm & Foster, 1989; Panayotou, 1975) suggest that green plant area expansion might be due to the growth stage at infection. Any such differentiation, not accounted for, among infections at different growth stages in the effect of BYDV on green plant area would be transferred to the calibrated RUE values.

The decreased allocation of biomass to grains is, as for the decreased green plant area, probably a result of damaged phloem tissue, which reduces the ability to transport photo-assimilates to grains. These results could be associated with the higher nitrogen concentrations found in the grains from BYDV-infected plants in the field experiment from 2002. The increased nitrogen concentrations might be caused by a decreased starch filling in infected plants, as previously claimed (Jensen & D'Arcy, 1995) to be the reason for similar results in previous experiments (Edwards *et al.*, 2001; Fitzgerald & Stoner, 1967; Potter, 1980). The fact that not only the nitrogen concentration but also total nitrogen content increased after infection at late growth stages suggests that other factors linked to nitrogen uptake mechanisms could also have contributed to the results. One explanation (Paper II) might be an exhaustion of nitrogen in the vegetative parts, which in line with the negative relationship between nitrogen and carbon phloem transport in uninfected plants shown by Fernandez-Figuares *et al.* (2000). However, the higher nitrogen concentration in uninfected plants at maturation (Paper III) shows that at least under conditions prevailing in that experiment, BYDV infections do not entail such effects.

In total, the model calibration was a first attempt to quantify effects of BYDV on parameters central to plant growth and grain yield. A few modifications in the design of new experiments, based on the difficulties encountered in the calibration, would help reduce possible sources of error and increase the reliability in the calibrated values. Such improvements could for example be other methods of determining intercepted radiation, in order to allow differences due to the decreased size of infected plants to be assessed. In addition, more frequent measurements of above-ground plant biomass and measurements of green plant

area in the field experiment would have given information about possible phases of recovery from the disease. Moreover, new experiments with measurements of nitrogen concentrations in different parts of infected plants would provide information about the relationship between light intensity, nitrogen concentration and growth rate in BYDV-infected plants, information that hopefully would help explain the reasons for the discrepancy between the greenhouse and field calibration.

Model tests

A test of the ability of the model to comply with its principle objective of simulating weather influence on grain yield reductions in BYDV-infected cereals was carried out. Cultivar, virus isolate, experimental site and infection procedure, all factors considered of importance to the degree of grain yield reduction in BYDV-infected oats, were the same as in the calibration experiment. Nevertheless, the simulated relative grain yield in BYDV-infected plants was 25% higher than observed. Other factors than those kept identical to the calibration experiment must have caused the deviation between the simulation and the observation. As mentioned above, there are several possible errors in the model calibration that might have contributed to the deviation. However, also factors such as water and nitrogen availability, which were not taken into account in the model structure, might have differed between the calibration and test experiments and affected the plant response to BYDV infection. Sub-models describing crop response to water (Amir & Sinclair, 1991b; Jamieson *et al.*, 1998b; Porter, 1993) and nitrogen content (Jamieson & Semenov, 2000; Jamieson *et al.*, 1998b; Jamieson, Stone & Semenov, 2001; Porter, 1993; Sinclair & Amir, 1992) have previously been formulated and tested for healthy cereal crops. Applying a nitrogen sub-model to describe BYDV-infected oats could be based on the experimental results for nitrogen concentration discussed above. Decreased root growth in BYDV-infected cereals (Hoffman & Kolb, 1997) could be accounted for in field applications by factors reducing water and nitrogen uptake.

An even more advanced model including water and nitrogen dynamics would only be able to simulate growth responses and grain yields of BYDV-infected oats under predefined conditions, which would include one specified virus isolate and one single oat cultivar. The results of the statistical analysis suggest a large variation in relative grain yield reductions depending on B/CYDV-isolates, cultivars, plant growth stages at infection and length of infection period. The results are also in line with previous studies from single sites in which growth stage at infection (Doodson & Saunders, 1970; Endo & Brown, 1963; Goulart, Ohm & Foster, 1989; Oswald & Houston, 1953; Smith, 1967), oat cultivar infected (Comeau & Dubuc, 1978; Gray, Smith & Sorrells, 1994), and the virus species and isolate used for inoculation (Baltenberger, Ohm & Foster, 1987; Chay *et al.*, 1996; Endo & Brown, 1963; Gray, Smith & Altman, 1993) affected the degree of grain yield reduction in B/CYDV-infected oats. Of these factors, only the plant growth stage at infection was taken into account in the simulation model. Likewise, simulated grain yields differed considerably from observations in experiments where factors treated in the statistical analysis also differed from

those in the calibration experiment. These findings suggest that the results of previous experiments referred to above are not secondary effects of weather variations. The importance of infection period on B/CYDV effect on grain yield in the GLM-analysis (Table 3) is doubtful. The time needed to inoculate B/CYDV in Coast Black oats (*Avena byzantina*) is shorter (Power, Seaman & Gray, 1991) than the periods analysed. The differences found could be a result of other factors, *e.g*. vector behaviour that indirectly might have affected the infection differently in the experiments analysed. Whatever the reason for the result, it emphasizes the need for protocols and standards in experiments including artificial virus infections if these experiments are to be comparable.

Conclusions

The model calibration showed that the reductions in above-ground biomass in BYDV-infected plants could partly be explained by changes in parameters regulating the development of the leaf area. The remainder of the difference in above-ground biomass between infected and uninfected plants could be ascribed to differences in RUE. BYDV infections also affect plant nitrogen status and protein content in grains. However, effects of BYDV infections on green plant area regulation parameters and RUE did not differ much between plants subject to different nitrogen fertilisation levels. Under field conditions, weather influences in B/CYDV impact on grain yield seem to be less important than other factors such as cultivar and virus isolate.

Future research questions

Further model modifications and calibrations

To allow the applicability of the model to be expanded, factors tested statistically here and in other studies referred to above should be taken into consideration. Cultivar differences are normally pronounced even in healthy plants and growth parameters in the simulation model have been calibrated specifically to different cultivars (Jamieson *et al.*, 1998b). The number of commercially grown oat cultivars is limited. For example, in Sweden in the period 2004/2005, seed from about 11 oat cultivars was certified in quantities that indicate cultivation on a large scale (http://www.utsadeskontrollen.se, accessed January 2006). It would be feasible to calibrate the model for these cultivars separately by complementing the design of experiments aimed at testing the B/CYDV tolerance of oat cultivars (Baltenberger, Ohm & Foster, 1987; Comeau & Dubuc, 1976; Goulart, Ohm & Foster, 1989) with measurements of central model parameters. Moreover, breeding and cultivation of specific oat cultivars are linked to specific climates. This correlation would entail some type of additional connection between the environmental factors included in the model and the cultivation of different oats

cultivars. Identification of such a relationship would probably simplify cultivar specific calibrations.

The genetic variation in B/CYDV-isolates occurring naturally is large (Miller, Liu & Beckett, 2002). To relate effects of that variability to simulation models would be a most challenging task. Before performing practical experiments with oats infected with different isolates, it would probably be more feasible to first identify possible distribution patterns for virus variants with different degrees of virulence. According to Comeau & Haber (2004), moderate virulence of B/CYDV is a favourable trait for survival. Imposing too many deleterious effects on its host plant would be unfavourable for the competitiveness of a virus isolate. Comeau & Haber (2004) further claim that virulence can be related to growth conditions of the host plant, where favourable growth conditions would select for more virulent genotypes. This alleged relationship could be coupled to the growth model by letting the same factors that affect plant growth also affect BYDV virulence. However, also factors that cannot easily be related to plant growth conditions are important to the spread of specific virus genotypes. For example, virus-vector interactions are crucial to the spread of different virus species and isolates (Power & Gray, 1995). There are mechanisms in vector acquisition (Gildow, 1993) and, supposedly more influential, transmission from vector to plant (Gildow, 1982; Gildow & Gray, 1993; Gildow & Rochow, 1980; Power & Gray, 1995) that are specific for different aphid-virus combinations. However, it is difficult to draw any conclusions about how this specificity affects the virulence of BYD-causing isolates. Still another factor that influences the occurrence of different B/CYDV genotypes is the population dynamics of the vector species. Their occurrence is, at least partly, based on factors other than those determining the growth of the host plants. Winds can transport aphid vectors long distances (Irwin & Thresh, 1988; Wallin, Peters & Johnson, 1967), resulting in infections of plants growing under environmental conditions different from those prevailing where the aphids acquired the virus. Populations of vector species are regulated by still other factors such as the frequency of their natural enemies (Chiverton, 1986; Ekbom, Wiktelius & Chiverton, 1992; Vickerman & Wratten, 1979) and the occurrence of wind shelters such as hedges and woodlands (Vickerman & Wratten, 1979), which do not directly impact on plant growth. In conclusion, there does not seem to exist any easily identifiable pattern in the occurrence of virus genotypes related to plant growth. As long as such patterns cannot be discerned, model calibrations to different virus genotypes would only frame a very small part of the variation occurring naturally in B/CYDV. Consequently, the virus genotype factor would preferably be more thoroughly evaluated in an ecological context before starting to parameterise its effect in a plant growth model.

Adaptation of the model to other crops and pathogens

The structure of the growth model used in this study to simulate oats was originally applied to simulate wheat (Amir & Sinclair, 1991a; Jamieson *et al.*, 1998b). The impact of BYDV in the model tested on oats would be applicable to simulate growth and grain yield in infected wheat or other cereals without any major structural changes. Simulations of infections in different cereal species

would give information about factors that cause different grain yield reductions among crops (Pike, 1987). The model could also be calibrated against cereal crops infected with other pathogens that cause similar symptoms and types of impact on grain yield on their host plants as does BYDV.

Coupling to other disease epidemiological models

Simulation models used for forecasting purposes constitute a useful tool for decision making in practical farming. Introducing disease factors into the simulation model could increase the precision and efficiency in combating plant pests. The growth model presented here could consist a sub-model in a decision support system aimed at optimising measures, *e.g.* chemical treatments of aphid vectors against BYDV. Combining models that simulate spread of BYDV among and within fields (Chaussalet *et al.*, 2000; Kendall, Brain & Chinn, 1992) with the model presented here aimed at assessing BYDV impact in infected plants would provide more precise forecasts on yield reduction than models only taking into account spread of the disease.

However, to be able to accurately simulate and forecast plant growth and grain yields, large amounts of resources are needed. Growth models often have a limited applicability concerning the range of environmental conditions under which they can predict total above-ground biomass and grain yield (Jamieson *et al.*, 1998a). Accurate simulation of the grain yield of different cereal cultivars under different environmental conditions would involve frequent recalibrations. Because of this cumbersome work, the benefit of including still another capricious factor when simulating disease in cereals could be questioned. Nevertheless, various diseases frequently attack and alter growth patterns of cereals, with subsequent yield losses. The range of applications of growth models would increase if disease factors were also considered.

Including disease factors when simulating cereal growth under future climate scenarios

Simulating growth and yield in future climate scenarios is another objective that weather-driven cereal models have focused on during recent years (Harrison, Butterfield & Downing, 2000). Predictions of plant growth and yield in future climate scenarios show changes in cereal productivity in Europe (Harrison, Butterfield & Orr, 2000; Olesen & Bindi, 2002). Climate changes are claimed to be partly the result of human activity (Rummukainen, 2005). The research area around simulating crop growth in a changed climate can be localised to the activity of avoiding or alleviating the most severe effects of climate changes. In this context, it should be mentioned that plant pests, similarly to what is alleged about climate change, are a phenomenon largely created by human activity. In wild plants, the net interactions between organisms considered as disease agents and their host can be both parasitic and mutualistic (Jarosz & Davelos, 1995). Accordingly, changes in the disease severity of many pathogens have paralleled changes in farming patterns and practices (Agrios, 1997). Experimental results also show that the level of atmospheric carbon dioxide affects the growth response

of oats to BYDV infections (Malmstrom & Field, 1997). Such effects suggest that simulating the growth of diseased crops under future climate scenarios would be a measure to forecast the future need for pest control.

References

Agrios, G.N. 1997. *Plant Pathology*. 4th edition. Academic Press. San Diego. 635 pp.

Amir, J. & Sinclair, T.R. 1991a. A model of the temperature and solar-radiation effects on spring wheat growth and yield. *Field Crops Research 28*, 47-58.

Amir, J. & Sinclair, T.R. 1991b. A Model of Water Limitation on Spring Wheat Growth and Yield. *Field Crops Research 28*, 59-70.

Andrews, C.J. & Paliwal, Y.C. 1983. The influence of preinfection cold hardening and disease development period on the interaction between barley yellow dwarf virus and cold stress tolerance in wheat. *Canadian Journal of Botany 61*, 1935-1940.

Baltenberger, D.E., Ohm, H.W. & Foster, J.E. 1987. Reactions of oat, barley, and wheat to infection with barley yellow dwarf virus isolates. *Crop Science 27*, 195-198.

Bauske, E.M., Bissonnette, S.M. & Hewings, A.D. 1997. Yield loss assessment of barley yellow dwarf disease on spring oat in Illinois. *Plant Disease 81*, 485-488.

Bisnieks, M., Kvarnheden, A., Sigvald, R. & Valkonen, J.P.T. 2004. Molecular diversity of the coat protein-encoding region of *Barley yellow dwarf virus-PAV* and *Barley yellow dwarf virus-MAV* from Latvia and Sweden. *Archives of Virology 149*, 843-853.

Bisnieks, M., Persson, T., Eckersten, H. & Sigvald, R. 2005. The effects on yield and components of yield in oats infected with BYDV-PAV at different growth stages. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 112*, 521-528.

Boote, K.J., Jones, J.W., Mishoe, J.W. & Berger, R.D. 1983. Coupling Pests to Crop Growth Simulators to Predict Yield Reductions. *Phytopathology 1581-1587*

Brooking, I.R., Jamieson, P.D. & Porter, J.R. 1995. The influence of daylength on final leaf number in spring wheat. *Field Crops Research 41*, 155-165.

Brouquisse, R., Masclaux, C., Feller, U. & Raymond, P. 2001. Protein Hydrolysis and Nitrogen Remobilisation in Plant Life and Senescence. In *Plant Nitrogen*. Edited by P.J. Lea & J.-F. Morot. Springer. Berlin. 407. pp.

Burnett, P.A., Comeau, A. & Qualset, C.O. 1995. Host Plant Tolerance of Resistance for Control of Barley Yellow Dwarf. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 321-343. pp.

Carrigan, L.L., Ohm, H.W., Foster, J.E. & Patterson, F.L. 1981. Response of winter wheat cultivars to barley yellow dwarf virus infection. *Crop Science 21*, 377-380.

Chaussalet, T.J., Mann, J.A., Perry, J.N. & Francos, R.J.C. 2000. A nearest neighbour approach to the simulation of spread of barley yellow dwarf virus. *Computers and Electronics in Agriculture 28*, 51-65.

Chay, C.A., Smith, D.M., Vaughan, R. & Gray, S.M. 1996. Diversity among isolates within the PAV serotype of barley yellow dwarf virus. *Phytopathology 86*, 370-377.

Chiverton, P.A. 1986. Predator density manipulation and its effects on populations of Rhopalosiphum padi (Hom.: Aphididae) in spring barley. *Annals of Applied Biology 109*, 49-60.

Comeau, A. 1987. Effects of BYDV inoculations at various dates in oats, barley, wheat, rye and triticale. *Phytoprotection 68*, 97-109.

Comeau, A. & Dubuc, J.P. 1976. Niveau de resistance de quelques cultivars d'avoine au virus du nanisme jaune de l'orge (BYDV). *Canadian Journal of Plant Science 56*, 397-398.

Comeau, A. & Dubuc, J.P. 1978. Evaluation of new and old sources of tolerance to early-season barley yellow dwarf virus in oats. *Canadian Journal of Plant Science 58*, 875-878.

Comeau, A. & Haber, S. 2004. Resistance and tolerance to BYDVs/CYDVs in cereals. In *Viruses and virus diseases of Poaceae (Gramineae)*. Edited by H. Lapierre & P.-A. Signoret. INRA Éditions. Paris. 204-218. pp.

Comeau, A. & St Pierre, C.A. 1975. Effets d'une infection hative vs. tardive des virus du nanisme jaune de l'orge (BYDV) sur des cultivars d'orge (Hordeum vulgare) avec vs. sans gene Yd2. *Canadian Journal of Plant Science 55*, 919-925.

D'Arcy, C.J. 1995. Symptomatology and Host Range of Barley Yellow Dwarf. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 9-28. pp.

Djurle, A. & Yuen, J.E. 1991. A simulation model for Septoria nodorum in winter wheat. *Agricultural Systems 37*, 193-218.

Doodson, J.K. & Saunders, P.J.W. 1970. Some effects of barley yellow dwarf virus on spring and winter cereals in field trials. *Annals of Applied Biology 66*, 361-374.

Eckersten, H. & Jansson, P.-E. 1991. Modelling water flow, nitrogen uptake and production for wheat. *Fertilizer Research 27*, 313-329.

Edwards, M.C., Fetch, T.G., Jr., Schwartz, P.B. & Steffenson, B.J. 2001. Effect of Barley yellow dwarf virus infection on yield and malting quality of barley. *Plant Disease 85*, 202-207.

Ekbom, B.S., Wiktelius, S. & Chiverton, P.A. 1992. Can polyphagous predators control the bird cherry-oat aphid (Rhopalosiphum padi) in spring cereals? A simulation study. *Entomologia Experimentalis et Applicata 65*, 215-223.

Endo, R.M. & Brown, C.M. 1963. Effects of Barley Yellow Dwarf Virus on Yield of Oats as Influenced by Variety, Virus Strain, and Developmental Stage of Plants at Inoculation. *Phytopathology 53*, 965-968.

Esau, K. 1957. Phloem degeneration in gramineae affected by the barley yellow dwarf virus. *American Journal of Botany 44*, 245-251.

Fernandes-Figares, I., Marinetto, J., Royo, C., Ramos, J.M. & Garcia del Morales, L.F. 2000. Amino-acid composition and protein and carbohydrate accumulation in the grain of triticale grown under terminal water stress simulated by a senescing agent. *Journal of Cereal Science 32*, 249-258.

Fitzgerald, P.J. & Stoner, W.N. 1967. Barley Yellow Dwarf Studies in Wheat (*Triticum aestivum* L.) I. Yield and quality of Hard Red Winter Wheat Infected With Barley yellow Dwarf Virus. *Crop Science 7*, 337-341.

Forde, S.M.D. 1993. Effects of temperature on BYDV concentration in oat plants and virus purification efficiency, and detection of a putative associated satellite virus. *Journal of Phytopathology 139*, 347-356.

Gildow, F.E. 1982. Coated-vesicle transport of luteoviruses through salivary glands of *Myzus persicae*. *Phytopathology 72*, 1289-1296.

Gildow, F.E. 1993. Evidence for receptor-mediated endocytosis regulating luteovirus acquisition by aphids. *Phytopathology 83*, 270-277.

Gildow, F.E. & Frank, J.A. 1988. Barley yellow dwarf virus in Pennsylvania: effect of the PAV isolate on yield components of noble spring oats. *Plant Disease 72*, 254-256.

Gildow, F.E. & Gray, S.M. 1993. The aphid salivary gland lamina basal as a selective barrier associated with vector-specific transmission of barley yellow dwarf luteovirus. *Phytopathology 83*, 1293-1302.

Gildow, F.E. & Rochow, W.F. 1980. Role of accessory salivary glands in aphid transmission of barley yellow dwarf virus. *Virology 104*, 97-108.

Gill, C.C. 1969. Annual variation in strains of barley yellow dwarf virus in Manitoba, and the occurence of greenbug-specific isolates. *Canadian Journal of Botany 47*, 1277-1283.

Gill, C.C. & Buchanon, K.W. 1972. Reactions of barley hybrids from crosses with C.I. 5791 to four isolates of barley yellow dwarf virus. *Canadian Journal of Plant Science 52*, 305-309.

Goulart, L.R., Ohm, H.W. & Foster, J.E. 1989. Barley yellow dwarf symptom severity in oat affected by plant growth stage at infection and plot type. *Crop Science 29*, 1412-1416.

Gourmet, C., Kolb, F.L., Smyth, C.A. & Pedersen, W.L. 1996. Use of imidacloprid as a seed-treatment insecticide to control barley yellow dwarf virus (BYDV) in oat and wheat. *Plant Disease 80*, 136-141.

Gray, S.M., Bergstrom, G.C., Vaughan, R., Smith, D.M. & Kalb, D.W. 1996. Insecticidal control of cereal aphids and its impact on the epidemiology of the barley yellow dwarf luteoviruses. *Crop Protection 15*, 687-697.

Gray, S.M., Smith, D. & Altman, N. 1993. Barley yellow dwarf virus isolate-specific resistance in spring oats reduced virus accumulation and aphid transmission. *Phytopathology 83*, 716-720.

Gray, S.M., Smith, D. & Sorrells, M. 1994. Reduction of disease incidence in small field plots by isolate-specific resistance to barley yellow dwarf virus. *Phytopathology 84*, 713-718.

Grindlay, D.J.C. 1997. Towards an explanation of crop nitrogen demand based on the optimization of leaf nitrogen per unit leaf area. *Journal of Agricultural Science 128*, 377-396.

Harrington, R. 2002. BYDV: The heat is on. In *Barley yellow dwarf disease: Recent advances and future strategies.*, Edited by M.M. Henry, A. Mexico: CIMMYT. 34-39. pp.

Harrison, P.A., Butterfield, R.E. & Downing, T.E. 2000. The CLIVARA project: Study aims and methods. In *Climate Change, Climate Variability and Agriculture in Europe-An Integrated Assessment*. Edited by T.E. Downing, P.A. Harrison, R.E. Butterfield & K.G. Lonsdale. Environmental Change Institute, University of Oxford. Oxford, UK. 3-10. pp.

Harrison, P.A., Butterfield, R.E. & Orr, J.L. 2000. Modelling climate change impacts on wheat, potato and grapevine in Europe. In *Climate Change, Climate Variability and Agriculture in Europe-An Integrated Assessment*. Edited by T.E. Downing, P.A. Harrison, R.E. Butterfield & K.G. Lonsdale. Environmental Change Institute, University of Oxford. Oxford, UK. 367-390. pp.

Herbert, D.A., Jr., Stromberg, E.L., Chappell, G.F. & Malone, S.M. 1999. Reduction of yield components by barley yellow dwarf infection in susceptible winter wheat and winter barley in Virginia. *Journal of Production Agriculture 12*, 105-109.

Hirose, T. & Werger, M.J.A. 1987. Nitrogen use efficiency in instantaneous and daily photosynthesis of leaves in the canopy of a *Solidago altissima* stand. *Physiologia Plantarum 70*, 215-222.

Hoffman, T.K. & Kolb, F.L. 1997. Effects of barley yellow dwarf virus on root and shoot growth of winter wheat seedlings grown in aeroponic culture. *Plant disease 81*, 497-500.

Hoffman, T.K. & Kolb, F.L. 1998. Effects of barley yellow dwarf virus on yield and yield components of drilled winter wheat. *Plant Disease 82*, 620-624.

Hull, R. 2002. *Matthews' Plant Virology*. 4th edition. Academic Press. London. 1002 pp.

Huth, W. 1993. Investigations on the behaviour of some winter barley cultivars after inoculation with barley yellow dwarf virus (BYDV). *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz 100*, 371-378.

Irwin, M.E. & Thresh, J.M. 1988. Long-range aerial dispersal of cereal aphids as virus vectors in North America. *Philosophical Transactions of the Royal Society of London B Biological Sciences 321*, 421-446.

Irwin, M.E. & Thresh, J.M. 1990. Epidemiology of barley yellow dwarf: a study in ecological complexity. *Annual Review of Phytopathology 28*, 393-424.

Jamieson, P.D., Porter, J.R., Goudriaan, J., Ritchie, J.T., Keulen, H.v. & Stol, W. 1998a. A comparison of the models AFRCWHEAT2, CERES-wheat, Sirius, SUCROS2 and SWHEAT with measurements from wheat grown under drought. *Field Crops Research 55*, 23-44.

Jamieson, P.D. & Semenov, M.A. 2000. Modelling nitrogen uptake and redistribution in wheat. *Field Crops Research 68*, 21-29.

Jamieson, P.D., Semenov, M.A., Brooking, I.R. & Francis, G.S. 1998b. Sirius: a mechanistic model of wheat response to environmental variation. *European Journal of Agronomy 8*, 161-179.

Jamieson, P.D., Stone, P.J. & Semenov, M.A. 2001. Towards modelling quality in wheat-from grain nitrogen concentration to protein composition. In *Aspects of Applied Biology 64, Wheat Quality*. Reading, UK: The Association of Applied Biologists. 111-126. pp.
Jarosz, A.M. & Davelos, A.L. 1995. Effects of disease in wild plant populations and the evolution of pathogen aggressiveness. *New Phytologist 129*, 371-387.
Jedlinski, H. 1972. Tolerance to Two Strains of Barley Yellow dwarf Virus in Oats. *Plant Disease Reporter 56*, 230-234.
Jenkyn, J.F. & Plumb, R.T. 1983. Effects of fungicides and insecticides applied to spring barley sown on different dates in 1976-79. *Annals of Applied Biology 102*, 421-433.
Jensen, S.G. 1969. Composition and metabolism of barley leaves infected with barley yellow dwarf virus. *Phytopathology 59*, 1694-1698.
Jensen, S.G. 1972. Metabolism and carbohydrate composition in barley yellow dwarf virus-infected wheat. *Phytopathology 62*, 587-592.
Jensen, S.G. & D'Arcy, C.J. 1995. Effects of Barley Yellow Dwarf on Host Plants. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 55-74. pp.
Jensen, S.G. & Van Sambeek, J.W. 1972. Differential effects of barley yellow dwarf virus on the physiology of tissues of hard spring wheat. *Phytopathology 62*, 290-293.
Kendall, D.A., Brain, P. & Chinn, N.E. 1992. A simulation model of the epidemiology of barley yellow dwarf virus in winter sown cereals and its application to forecasting. *Journal of Applied Ecology 29*, 414-426.
Lapierre, H., Becket, R., Miller, W.A. & Jacquot, E. 2004. Taxa of viruses, virus-like and subviral agents infecting *Poaceae* Family *LUTEOVIRIDAE*. In *Viruses and virus diseases of Poaceae (Gramineae)*. Edited by H. Lapierre & P.-A. Signoret. INRA Éditions. Paris. 366-374. pp.
Lawlor, D.W., Lemaire, G. & Gastal, F. 2001. Nitrogen, Plant Growth and Crop Yield. In *Plant Nitrogen*. Edited by P.J. Lea & J.-F. Morot. Springer-Verlag. Berlin. 343-367. pp.
Leclercq-Le Quillec, F., Plantegenest, M., Riault, G. & Dedryver, C.A. 2000. Analyzing and modeling temporal disease progress of Barley yellow dwarf virus serotypes in barley fields. *Phytopathology 90*, 860-866.
Lister, R.M. & Ranieri, R. 1995. Distribution and Economic Importance of Barley Yellow Dwarf. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 29-53. pp.
Lowe, A.D. 1967. Avoid yellow dwarf virus by late sowing. *New Zealand Wheat Review 10*, 59-63.
Luo, Y., Teng, P.S., Fabellar, N.G. & TeBeest, D.O. 1997. A rice-leaf blast combined model for simulation of epidemics and yield loss. *Agricultural Systems 53*, 27-39.
Malmstrom, C.M. & Field, C.B. 1997. Virus-induced differences in the response of oat plants to elevated carbon dioxide. *Plant, Cell and Environment 20*, 178-188.
McCown, R.L., Hammer, G.L., Hargreaves, J.N.G., Holzworth, D.P. & Freebairn, D.M. 1996. APSIM: a novel software system for model development, model testing and simulation in agricultural systems research. *Agricultural Systems 50*, 255-271.
McElhany, P., Real, L.A. & Power, A.G. 1995. Vector preference and disease dynamics: a study of barley yellow dwarf virus. *Ecology 76*, 444–457.
McGrath, P.F. & Bale, J.S. 1990. The effects of sowing date and choice of insecticide on cereal aphids and barley yellow dwarf virus epidemiology in northern England. *Annals of Applied Biology 117*, 31-43.
Miller, W.A., Liu, S.J. & Beckett, R. 2002. Barley yellow dwarf virus: Luteoviridae or Tombusviridae? *Molecular Plant Pathology 3*, 177-183.
Monneveux, P., St Pierre, C.A., Comeau, A. & Makkouk, K.M. 1992. Barley yellow dwarf virus tolerance in drought situations. In *Barley yellow dwarf in West Asia and North Africa*, Edited by A. Comeau & K.M. Makkouk: International Centre for Agricultural Research in the Dry Areas; Aleppo; Syria. 209-220. pp.
Monteith, J.L. & Unsworth, M.L. 1990. *Principles of environmental physics*. Second edition. Edward Arnold. London. 291 pp.

Niks, R.E., Habekuss, A., Bekele, B. & Ordon, F. 2004. A novel major gene on chromosome 6H for resistance of barley against the barley yellow dwarf virus. *Theoretical and Applied Genetics 109*, 1536-1543.
Olesen, J.E. & Bindi, M. 2002. Consequences of climate changes for European agricultural productivity, land use and policy. *European Journal of Agronomy 16*, 239-262.
Orlob, G.B. & Arny, D.C. 1961. Some metabolic changes accompanying infection by barley yellow dwarf virus. *Phytopathology 51*, 768-775.
Oswald, J.W. & Houston, B.R. 1953. The Yellow-Dwarf virus Disease of Cereal Crops. *Phytopathology 43*, 128-136.
Paliwal, Y.C. & Andrews, C.J. 1979. Effects of barley yellow dwarf and wheat spindle streak mosaic viruses on cold hardiness of cereals. *Canadian Journal of Plant Pathology 1*, 71-75.
Panayotou, P. (1975). *Barley yellow dwarf virus in cereals and grasses,* University of Exeter.
Panayotou, P. 1978. Degenerative changes induced by barley yellow dwarf virus on certain tissues of several cereals. *Phytopathologia Mediterranea 17*, 23-29.
Peltonen-Sainio, P., Forsman, K. & Poutala, T. 1997. Crop Management Effects on Pre- and Post- Anthesis Changes in Leaf Area Index and Leaf Area Duration and their Contribution to Grain Yield and Yield Components in Spring Cereals. *Journal of Agronomy and Crop Science 179*, 47-61.
Persson, T., Eckersten, H., Kvarnheden, A. & Yuen, J. 2006. Modelling influence of virus infection on leaf area and radiation use in oats under controlled climatic conditions. *Acta Agriculturae Scandinavica Section B-Soil and Plant In press*
Pike, K.S. 1987. A review of barley yellow dwarf virus grain yield losses. In *World Perspectives on Barley Yellow Dwarf International Workshop*. Udine Italy. 356-361. pp.
Pinnschmidt, H., Teng, P.S. & Yuen, J. 1990. Pest Effects on Crop Growth and Yield. In *Workshop on Modeling Pest-crop Interactions*, Edited by P.S. Teng & J. Yuen. Honolulu, USA. 26-29. pp.
Plumb, R.T. 1992. Barley yellow dwarf. In *Plant diseases of international importance.* Edited by U.S. Singh, A.N. Mukhopadhyay, J. Kumar & H.S. Chaube. Prentice Hall Inc. Englewood Cliffs, New Jersey; USA. 41-79. pp.
Plumb, R.T. & Johnstone, G.R. 1995. Cultural, Chemical, and Biological Methods for the Control of Barley Yellow Dwarf. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 307-319. pp.
Porter, J.R. 1984. A model of canopy development in winter wheat. *Journal of Agricultural Science 102*, 383-392.
Porter, J.R. 1985. Models and mechanisms in the growth and development of wheat. *Outlook on Agriculture 14*, 190-196.
Porter, J.R. 1993. AFRCWHEAT2: A model of the growth and development of wheat incorporating responses to water and nitrogen. *European Journal of Agronomy 2*, 69-82.
Porter, J.R., Jamieson, P.D. & Wilson, D.R. 1993. Comparison of the wheat simulation models AFRCWHEAT2, CERES-Wheat and SWEAT for non-limiting conditions of crop growth. *Field Crops Research 33*, 131-157.
Potter, L.R. 1980. The effects of barley yellow dwarf virus and powdery mildew in oats and barley with single and dual infections. *Annals of Applied Biology 94*, 11-17.
Power, A.G. & Gray, S.M. 1995. Aphid Transmission of Barley Yellow Dwarf Viruses: Interactions Between Viruses, Vectors, and Host Plants. In *Barley Yellow Dwarf 40 Years of Progress*. Edited by C.J. D'Arcy & P.A. Burnett. The American Phytopathological Society. St. Paul, MN. 259-289. pp.
Power, A.G., Seaman, A.J. & Gray, S.M. 1991. Aphid transmission of barley yellow dwarf virus: inoculation access periods and epidemiological implications. *Phytopathology 81*, 545-548.
Rabbinge, R., Jorritsma, I.T.M. & Schans, J. 1985. Damage components of powdery mildew in winter wheat. *Netherlands Journal of Plant Pathology 91*, 235-247.
Rasmusson, D.C. & Schaller, C.W. 1959. The inheritance of resistance in barley to the yellow-dwarf virus. *Agronomy Journal 51*, 661-664.

Ritchie, J.T. & Otter, S. 1985. Description and performance of CERES-wheat. A user-oriented wheat yield model ARS wheat yield project. *ARS U S Department of Agriculture, Agricultural Research Service*, 159-176.
Rochow, W.F. 1969. Biological Properties of Four Isolates of Barley Yellow Dwarf Virus. *Phytopathology 59*, 1580-1589.
Rochow, W.F. & Muller, I. 1971. A Fifth Variant of Barley Yellow Dwarf Virus in New York. *Plant Disease Reporter 55*, 874-877.
Rouse, D.I. 1988. Use of Crop Growth-Models to Predict the Effects of Disease. *Annual Review of Phytopathology 26*, 183-201.
Rummukainen, M. (2005). Växthuseffekten. 119. SMHI-Swedish Meteorological and Hydrological Institute.
Shepherd, R.J., Francki, R.I.B., Hirth, L., Hollings, M., Inouye, T., MacLeod, R., Purcifull, D.E., Sinha, J.H., Tremaine, J.H., Valenta, V. & Wetter, C. 1976. New Groups of Plant Viruses Approved by the International Commitee on Taxonomy of Viruses, September 1975. *Intervirology 6*, 181-184.
Sinclair, T.R. & Amir, J. 1992. A Model to Assess Nitrogen Limitations on the Growth and Yield of Spring Wheat. *Field Crops Research 30*, 63-78.
Sinclair, T.R. & Muchow, R.C. 1999. Radiation Use Efficiency. *Advances in Agronomy 65*, 215-265.
Skaria, M., Lister, R.M. & Foster, J.E. 1984. Lack of barley yellow dwarf virus dosage effects on virus content in cereals. *Plant Disease 68*, 759-761.
Skaria, M., Lister, R.M., Foster, J.E. & Shaner, G. 1985. Virus content as an index of symptomatic resistance to barley yellow dwarf virus in cereals. *Phytopathology 75*, 212-216.
Slykhuis, J.T., Zillinsky, F.J., Hannah, A.E. & Richards, W.R. 1959. Barley Yellow Dwarf Virus on Cereals in Ontario. *Plant Disease Reporter 43*, 849-854.
Smith, H.C. 1967. The Effect of aphid Numbers and Stage of Plant Growth in Determining Tolerance to Barley Yellow Dwarf Virus in Cereals. *New Zealand Journal of Agricultural Research 10*, 445-466.
Sogaard, B. & von Wettstein-Knowles, P. 1987. Barley: genes and chromosomes. *Carlsberg Research Communications 52*, 123-196.
Sonego, M., Moot, D.J., Jamieson, P.D., Martin, R.J. & Scott, W.R. 2000. Apical development in oats predicted by leaf stage. *Field Crops Research 65*, 79-86.
Wallin, J.R., Peters, D. & Johnson, L.C. 1967. Low-Level Jet winds, Early Cereal Aphid and Barley Dwarf Detection in Iowa. *Plant Disease Reporter 51*, 527-530.
van der Werf, W. (1988). *Yellowing viruses in sugarbeet; epidemiology and damage,* Landbouwuniversiteit te Wageningen.
Watson, M.A. & Mulligan, T.E. 1960. Comparison of two barley yellow-dwarf viruses in glasshouse and field experiments. *Annals of Applied Biology 48*, 559-574.
Weir, A.H., Bragg, L.P., Porter, J.R. & Rayner, J.H. 1984. A winter wheat crop model without water or nutrient limitation. *Journal of Agricultural Science 102*, 371-382.
Vickerman, G.P. & Wratten, S.D. 1979. The biology and pest status of cereal aphids (Hemiptera: Aphididae) in Europe: a review. *Bulletin of Entomological Research 69*, 1-32.
Zadoks, J.C., Chang, T.T. & Konzak, C.F. 1974. A decimal code for the growth stages of cereals [Maize, sorghum, forage grasses and dicotyledonous crops]. *Weed Research 14*, 415-421.
Zhang, Q.F., Guan, W.N., Ren, Z.Y., Zhu, X.S. & Tsai, J.H. 1983. Transmission of barley yellow dwarf virus strains from northwestern China by four aphid species. *Plant Disease 67*, 895-899.

Acknowledgements

The financial support of this thesis came from the SLU-faculty theme *Effects of plant production systems on growth and product quality*, and also from C. F. Lundströms stiftelse and E. H. Hügoths stiftelse.

I thank my main supervisor Henrik Eckersten for help, support and encouragement; and for many interesting and inspiring discussions about modelling. I also thank my supervisors Anders Kvarnheden and Jonathan Yuen for help and support in the work with Paper III and IV and for coming up with questions from other research fields linked to BYDV such as virology and epidemiology.

I also thank Maris Bisnieks for co-operation with Paper I and the experimental work it was based on, Annhild Andersson for instructions and help with ELISA tests and aphid cultivation, and Roland Sigvald for letting me use data from the field experiment in 2003 in my thesis work.

I thank CG Pettersson for interesting talks and discussions in our shared office and, Johan Ahnström, Maria Björkman, Eva Blixt, Hanna Friberg, Oscar Gomez, Anushka Heeb, Hasna Mahbuba Kaniz, Sandra Pye, Francisco Salmeron, Johanna Sjöberg, Alireza Taab, Maria Viketoft and Liv Åkerblom Espeby for interesting discussions about questions shared by Ph. D. students.

I thank Carolyn Malmstrom, Todd Robinson, Toshi Yoshida, Scott Butterfield**,** Abbie Schrotenboer and Jennie Shu for giving me the great opportunity to spend a couple of months at your laboratory at Michigan State University.

I thank Per Nyman for providing weather data and help with computer problems.

I thank Göran Bergkvist and Lars Andersson for, as supervisors in my examensarbete, inspiring me to continue my education with Ph. D. studies.

I thank Mary McAfee for proof reading my English.

I thank Sadhna Alström for advices and encouragement in the planning of my Ph.D. studies.

I thank other people at the former department of Ecology and Crop Production Science, especially Anna-Karin Widmark for advices and discussions about aphid cultivations and laboratory practices, Stina Carlsson, Lena Färeby, Sven-Erik Karlsson, Henrik af Geijerstam, Jan-Olof Pettersson, K-G Ursberg, Carl Åkerberg, Berit Lundén, Gunilla Alfons, and Birgitta Bergstrand-Holm for help with different technical, laboratory or administrative questions.

I thank my parents, brothers and sister-in-law, and other friends for support and encouragements.