Crop architecture and crop tolerance to fungal diseases and insect herbivory. Mechanisms to limit crop losses

Article *in* European Journal of Plant Pathology · November 2012

DOI: 10.1007/s10658-012-0125-z

CITATIONS
21

READS
314

8 authors, including:

Bertrand Ney
MINES ParisTech
84 PUBLICATIONS **2,792** CITATIONS
SEE PROFILE

Marie-Odile Bancal
French National Institute for Agricultural Research
63 PUBLICATIONS **594** CITATIONS
SEE PROFILE

Pierre Bancal
French National Institute for Agricultural Research
34 PUBLICATIONS **497** CITATIONS
SEE PROFILE

Ian Bingham
Scotland's Rural College
52 PUBLICATIONS **1,595** CITATIONS
SEE PROFILE

Eur J Plant Pathol
DOI 10.1007/s10658-012-0125-z

SPECIAL ISSUE 'ARCHITECTURE AND EPIDEMIOLOGY'

Crop architecture and crop tolerance to fungal diseases and insect herbivory. Mechanisms to limit crop losses

B. Ney · M. O. Bancal · P. Bancal · I. J. Bingham · J. Foulkes · D. Gouache · N. Paveley · J. Smith

Accepted: 5 November 2012

Abstract Plant tolerance to biotic stresses (mostly limited here to fungal pathogens and insects) is the ability of a plant to maintain performance in the presence of expressed disease or insect herbivory. It differs from resistance (the capacity to eliminate or limit pests and pathogens by genetic and molecular mechanisms) and avoidance (the ability to escape infection by epidemics). The ways to tolerance of pests and diseases are multiple and expressed at different scales. The contribution of organs to the capture and use of resources depends on canopy and root architecture, so the respective locations of disease and plant organs will have a strong effect on the crop's response. Similarly, tolerance is increased when the period of crop sensitivity lies outside the period within which the pest or pathogen is present. The ability of the plant to compensate for the reduced acquisition of resources by the production of new organs or by remobilization of reserves may also mitigate biotic stress effects. Numerous examples exist in the literature and are described in this article. Quantification of tolerance remains difficult because of: (i) the large number of potential mechanisms involved; (ii) different rates of development of plants, pests and pathogens; and (iii) various compensatory mechanisms. Modelling is, therefore, a valuable tool to quantify losses, but also to prioritize the processes involved.

Keywords Diseases · Insects · Tolerance · Crop architecture · Modelling

B. Ney (✉) · M. O. Bancal
AgroParisTech, UMR 1091 INRA-AgroParisTech Environnement et Grandes cultures,
78850 Thiverval-Grignon, France
e-mail: ney@agroparistech.fr

P. Bancal
INRA, UMR 1091 INRA-AgroParisTech Environnement et Grandes cultures,
78850 Thiverval-Grignon, France

I. J. Bingham
Crop & Soil Systems Research Group, SRUC,
West Mains Rd.,
Edinburgh, UK

J. Foulkes
Division of Plant and Crop Sciences, School of Biosciences,
University of Nottingham,
Sutton Bonington campus,
Loughborough, UK LE12 5RD

D. Gouache
ARVALIS - Institut du Végétal, Service Génétique Physiologie et Protection des Plantes, IBP - Université Paris Sud,
Rue de Noetzlin - Bât. 630,
91405 Orsay Cedex, France

N. Paveley · J. Smith
ADAS UK Ltd, Sustainable Crop Management,
Rosemaund, Preston Wynne,
Hereford, UK HR1 3PG

Definitions

The term "tolerance" has been widely used in ecology, pathology and agronomy in order to define the strategy by which plants, populations or communities are able to limit damage when directly exposed to abiotic and biotic stresses (Beattie and Lindow 1995). The term "damage"

Published online: 17 November 2012

means "injury" for many herbivory researchers but "yield loss" for others (Savary et al. 2006). Actually, tolerance is human- rather than plant-directed, and therefore damage can refer to any plant, population or community characteristic of interest. In many cases, the damage against which tolerance is measured is loss in grain yield, but tolerance in reference to ecological fitness or harvest quality could be emphasized as well, whatever measure of quality is used. Precise knowledge about what characteristic is damaged by stresses is necessary to identify the ways to tolerance.

However, the term "tolerance" has been used broadly and this has resulted in many meanings (Schafer 1971). Maintaining multiple interpretations of the definition of the term has led to confusion in the literature and there is a need to clarify the situation, particularly in the case of biotic stresses. Within the plant pathology field, the term tolerance has often been used to mean resistance (Kluth and Varrelmann 2010; Aguado et al. 2010), reduction in rate of pathogen development (Gao et al. 1995; Mussell and Malone 1979) and pathogen tolerance (Clarke 1984). Foulkes et al. (2006) restricted the definition to "the ability to maintain yield performance despite the presence of disease symptoms", while more general definitions were given by Oliver et al. (2009) stating that "tolerance traits minimize fitness loss of the actor but without reducing encounter rate" and Caldwell et al. (1958) who considered tolerance as a means by which plants endure severe disease without severe losses in yield or quality.

The frontiers of tolerance with resistance and avoidance, both of which serve to reduce the incidence or severity of attacks (Roy and Kirchner 2000), might be challenged. Indeed the ability of the host to accommodate a pathogen with reduced symptoms is close to a form of resistance (the capacity of plants to eliminate or limit pests and pathogen infection by genetic and molecular mechanisms). Similarly, a trait that limits the spread of a pest or pathogen within the canopy is regarded as an avoidance mechanism, but at the same time it may confer tolerance of a given severity of disease or herbivory if the location of the pathogen or pest is confined to non-essential organs. Such restrictions are in agreement with the definition that Schafer (1971) gave for tolerance as "the capacity of a cultivar resulting in less yield or quality loss relative to disease severity or pathogen development when compared with other cultivars or crops". This definition highlights two important points: (i) in order to quantify tolerance, a reference point must first be established against which other comparable plants or crops can be measured; and (ii) as restated by Clarke (1986) and Robb (2007), in the case of pathogens a distinction must be made between the tolerance of a host to the pathogen (i.e. the ability of the host to limit symptom expression under similar levels of infection) and the tolerance of a host to the symptoms caused by the pathogen (i.e. the ability of a host to limit physiological disruption under similar levels of disease expression). Both may coexist at the plant or the crop level. This distinction is critical especially when considering biotic injury which may not be expressed as visible symptoms, as in the case of certain viral infections (Robb 2007).

The definition of disease tolerance was further refined by Parker et al. (2004) who described it as "the heritable capacity of a crop to maintain productivity despite the presence of disease". These authors suggest that traits, which confer disease tolerance, are separate from those which confer resistance and thus may be manipulated independently (Roux et al. 2010). The literature also points out that environmental factors and fitness costs associated with tolerance may play a role in tolerance expression in pathogen-plant interactions, but this has been largely overlooked (Tiffin and Rausher 1999; Fornoni et al. 2004).

For herbivory, tolerance is defined as the ability a plant has to sustain a fixed amount of injury without a corresponding reduction in fitness (Mauricio et al. 1997); it is classed as a compensatory capacity because plant losses, rather than injury, are prevented. For viruses, Cooper and Jones (1983) defined tolerance as the absence of symptoms or the occurrence of only small symptoms, and a little or no loss in vigour or yield of infected plants. Symptoms are not necessarily correlated with virus concentration. The authors clearly separate resistance which involves decreasing virus concentration by lowering multiplication and/or invasion from tolerance which involves decreasing symptoms at a given concentration of virus.

In this paper we define tolerance to biotic stresses (confined here mainly to fungal diseases and insect herbivory) as the ability of a plant or a crop to maintain performance, fitness or a high quality characteristic in the presence of expressed symptoms (or significant concentration of virus), i.e. actual injury or disease. Thus the determinants of its variability within a species may be considered as a means with

which to manage the effects of diseases and pests on crop growth, yield and quality (Kover and Schaal 2002). It is anticipated that tolerance will complement, rather than replace, resistance and avoidance traits.

The ways to tolerance are diverse. In some cases, the disease or pest may be located on plant organs that are little involved in essential plant functions such as photosynthesis. In other cases, compensations may occur by the production of new healthy organs or enhancement of plant functions (photosynthesis, nutrients or water uptake). Tolerance in that case may relate to the capacity (or not) of the plant to modify its architecture (the spatial arrangement of its organs). Tolerance may also arise from variation in the time when disease or herbivory occurs. Some periods of the crop growth cycle are not sensitive to attacks, because sensitive organs have not yet appeared. Remobilization of plant reserves may also buffer the plant against the effects of a decrease in nutrient acquisition and photo-assimilate production. Therefore, in many cases plant architecture, as the organization of plant components in space, which can change with time (Godin et al. 1999), is involved in tolerance. In this paper, we show, with some examples, how plant architectural traits may be ways to tolerance, and how modelling can be used to link architectural traits to crop physiology and yield formation and thus help understand and quantify tolerance.

Overview of tolerance pathways to diseases and other pests

Yield determination and disease occurrence

The physiological processes of yield formation must be briefly considered in order to determine at which developmental stages the crop is likely to tolerate the effects of pathogens and pests. The decomposition of the yield into components has been successfully used to examine the processes involved in abiotic and biotic stress. Many works (Gallagher et al. 1975; Gambı´n et al. 2006) have decomposed grain crop yield into two main components: seed number m^{-2} (SN) and mean seed weight (MSW). Crop growth is related to the amount of intercepted photosynthetically active radiation (IPAR) and the efficiency (radiation use efficiency RUE) with which the solar energy is converted into dry matter (Reynolds et al. 2005; Bingham et al. 2007a). Pathogens may reduce yield by limiting the formation of any of the yield components and the majority of crops fall short of achieving their yield potential (Spink et al. 2000; Sylvester-Bradley and Wiseman 2005). Infected crops can become either source-limited (i.e. dry matter or nitrogen acquisition is the limiting factor) or sink-limited (i.e. the storage capacity of reproductive organs is less than the supply of assimilate to fill them), depending on the environmental conditions (edaphic and/or agronomic). For many crop species the storage capacity of the reproductive organs is a function of the number of seeds and their potential size. SN is generally closely correlated to the dry matter accumulated between emergence and flowering. It can be reduced either by direct pest attacks or by photosynthate shortage during some critical periods that may lead to a dramatic decrease of SN, because of uncompensated floret abortion (Fischer and Stockman 1980). In many production systems diseases and pests are then not only problematic during rapid grain filling, but also during the critical period when SN is determined. MSW, on the other hand, is often related to the rate of grain filling as governed by post-flowering assimilate availability. But here too there is evidence of sink-limitation as the carpel size and seed cell number, which may be genetically and environmentally dependant, set the storage capacity and thus potential seed size (Munier-Jolain and Ney 1998; Bingham et al. 2007b). Thus MSW may be reduced by disease or insect pests not only through their effects on the availability of assimilate for grain filling, but also earlier through the effects of reduced assimilate availability on carpel growth and seed number. In either case, the impact of pathogens and pests on MSW may be negated by the remobilization of previously accumulated storage reserves which can buffer a temporal lack of assimilates.

In conclusion, candidate traits determining tolerance will differ between pathosystems and will be influenced by whether the crop is source or sink limited and the extent to which pests and diseases constrain yield through crop dry matter source or sink components. Different crops will have different tolerances to similar pests and improved disease and pest management would be supported by a better knowledge of host × pest interactions at the crop level. The previous yield decomposition is helpful to estimate the

overall impact of pests on sources (by reduction of resource acquisition balanced by reserve mobilisation), sinks (by floret abortion balanced by new healthy organs appearance) and overall effects on partitioning (by competition between pathogen and host organs balanced by switches in source-sink relationships). The different mechanisms associated with tolerance to biotic stress operate at three levels (organ, plant, and canopy) that can be distinguished to enhance comprehension (Fig. 1).

Tolerance at organ level: the role of virtual lesion

The following examples mostly deal with net carbon assimilation in infected or grazed leaves, but tolerance also applies to both nitrogen acquisition by infected roots and biomass remobilisation by any kind of organ. Damage to leaf tissue through infection by foliar pathogens or insect herbivory can invoke complex changes in the plant's carbon metabolism and assimilate partitioning. The nature and extent of the response differs widely depending on the pathogen or herbivore in question, its mode of nutrition, the nature of the host-pathogen interaction (compatible or incompatible), the host species and in some cases variety, and the physiological status and age of the organ. Fungal pathogens can be classified as necrotrophic, biotrophic, or hemi-biotrophic according to their nutritional relationship with the host (see Oliver and Ipcho 2004, for definitions). Necrotrophs kill tissue in advance of colonisation by fungal hyphae through the secretion of toxins and cell wall degrading enzymes, whilst biotrophs derive their resources for growth and sporulation from living host cells. As such, biotrophs have a more subtle impact on photosynthetic metabolism, effectively altering source-sink relations within the leaf and directing host nutrients to the fungal mycelium (Scholes and Rolfe 2009; Bancal et al. 2012). Hemi-biotrophs

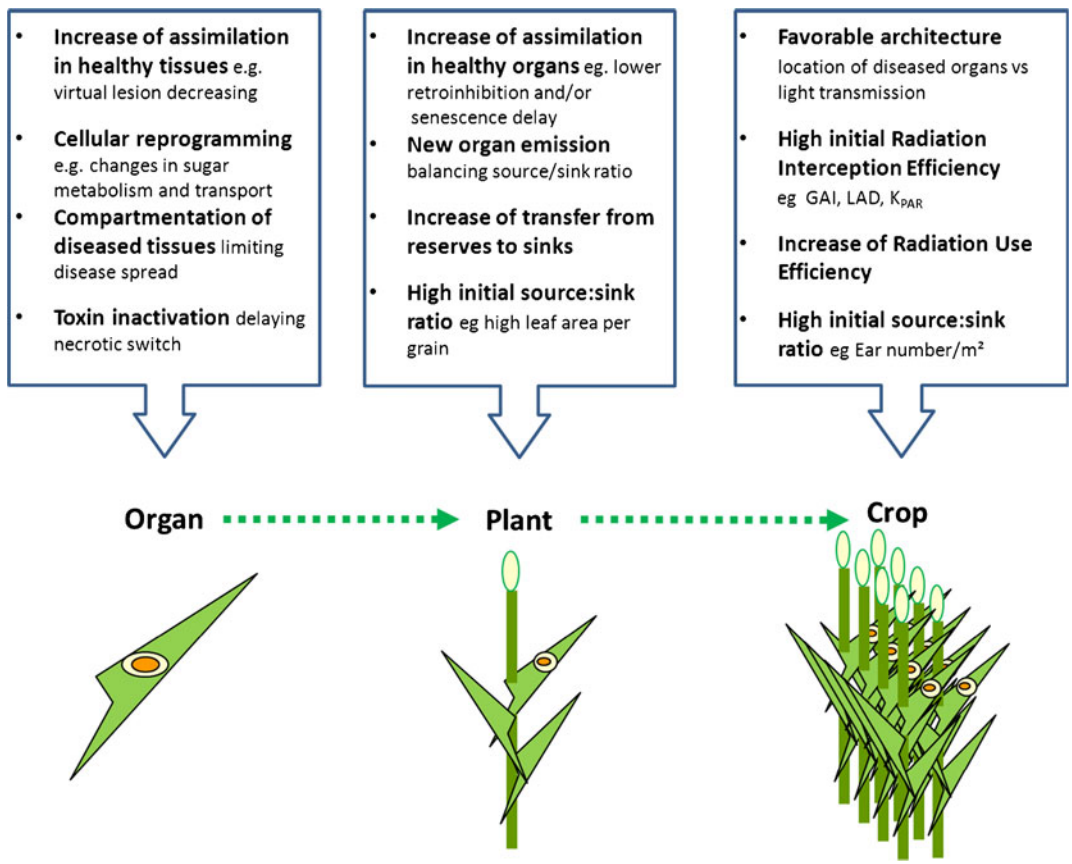

Fig. 1 Diagram presenting the three levels (organ-plant-crop) and the functions (traits) involved in tolerance at each level to highlight the potential research axes. Some tolerance traits are not influenced by crop architecture (intrinsic tolerance; e.g. virtual lesion) while other traits are influenced by plant and crop architecture (e.g. source-sink relationships)

behave initially as biotrophs, but then subsequently switch to a necrotrophic mode of nutrition.

When examined on a whole leaf basis, biotrophic and necrotrophic infection often leads to a reduction in net photosynthesis associated, to varying extents, with alterations in stomatal behaviour, chlorophyll concentrations, perturbations in the light reactions of photosynthesis and reductions in the amounts and activities of Benson-Calvin cycle enzymes. However, such whole leaf measurements mask considerable spatial and temporal heterogeneity in host response to infection, which could influence the host's apparent disease tolerance when tolerance is defined as the ability to sustain growth in the presence of visible disease. In principle low tolerance (relative intolerance) might occur where photosynthetic metabolism is disrupted or respiration increased before the appearance of visible symptoms, or where these effects extend into regions of green tissue beyond the visible lesion after symptoms develop. Conversely, increases in rates of photosynthesis in non-infected regions of infected leaves in response to the loss of photosynthetic activity elsewhere could result in the apparent tolerance of disease (Bingham et al. 2009).

It has been recognised for some years that the reduction in rate of net photosynthesis in infected leaves cannot always be explained quantitatively in terms of the area of visible lesions and the consequent loss of green tissue (Martin 1986; Shtienberg 1992). Bastiaans (1991) developed the concept of the virtual lesion to account for the effects of disease in asymptomatic regions of infected leaves and the concept has been widely used for modelling the effects of foliar disease on canopy photosynthesis and crop growth (Bastiaans and Kropff 1993; Garry et al. 1998; Robert et al. 2004; Bingham and Topp 2009). A virtual lesion is the area of the leaf within which the photosynthetic rate is negligible. A ratio of virtual lesion area to visible lesion area (β in Bastiaans' model, Bastiaans 1993b) greater than 1.0 implies that photosynthesis is inhibited in green tissue beyond the visible lesion. Values of β ranging from 1.3 to 12 have been reported for rust and powdery mildew infected cereals (Rossing et al. 1992; Robert et al. 2005; 2006), anthracnose infected bean (*Phaseolus vulgaris*) (Lopes and Berger 2001) or Ascochyta blight (caused by *Mycosphaerella pinodes*) infected pea (*Pisum sativum* L.) (Garry et al. 1998). For a given pathosystem, the β value can also differ depending on either host age or lesion development. Altogether, it suggests wide variation in the severity of inhibition within symptomless regions. However, the literature is complicated as the β value changes with the nature of the symptom assessed. Thus values were greatest when only the sporulating area of rust-infected wheat (*Triticum aestivum* L.) was included in the visible lesion and least when sporulating (Robert et al. 2005; 2006).

Chlorophyll fluorescence imaging allows the heterogeneous effects of pathogen infection on photosynthetic metabolism to be studied at a greater spatial resolution than is possible with gas exchange techniques (Scholes and Rolfe 2009; Rolfe and Scholes 2010). This approach has provided direct confirmation that photosynthesis can be modified in symptomless regions distant from the visible lesion, but that the nature and temporal dynamics of the response can differ markedly between pathosystems. For example, in oat (*Avena sativa*) leaves infected by the biotrophic pathogen *Puccinia coronata* the causal agent of crown rust, there was a small-localised reduction in photochemical efficiency (Φ_{II}) 5 days after inoculation in regions associated with fungal mycelium and the development of visible symptoms (Scholes and Rolfe 1996). However, non-photochemical energy dissipation (qN) was greatly reduced across the symptomless regions compared to non-inoculated controls suggesting an increase in demand for ATP within the leaf as a whole. Eight days after inoculation, coinciding with sporulation of the pathogen, Φ_{II} was reduced and qN increased in symptomless regions consistent with an inhibition of photosynthesis distant from the visible lesion. In other examples, such as *Arabidopsis thaliana* infected by the white blister rust (*Albugo candida,* a biotroph), effects on photosynthesis were confined to invaded tissue (Chou et al. 2000). Similar contrasts in response have been reported for necrotrophic and hemi-biotrophic pathosystems. *Colletotrichum lindemuthianum* (anthracnose) inhibited photosynthesis in regions between visible lesions in beans (Meyer et al. 2001), whilst increases in photosynthesis have been observed in symptomless regions of tomato (*Solanum lycopersicum*) leaves infected by *Botrytis cinerea* (grey mould) and wheat leaves infected by *Mycosphaerella graminicola* (Septoria leaf blotch) (Berger et al. 2004; Scholes and Rolfe 2009).

Rolfe and Scholes (2010) also pointed out that in addition to the lack of general rules regarding responses within the same fungal nutritional group, no consistent response has been found with pathogen type (virus, bacteria or fungi). However in the case of

viral- and bacterial-plant interactions, an increased rate of photosynthesis at the transition between symptoms and green parts has often been shown, and attributed to an increased demand for energy reflecting a high metabolic activity.

Leaf responses to insect herbivory are, in many respects, similar to those induced by fungal pathogens. Photosynthesis is often reduced to a greater extent than would be predicted by the loss of green leaf tissue, with rates in some regions of remaining healthy tissue being impaired (Zangerl et al. 2002; Nabity et al. 2009; Tang et al. 2009). A number of mechanisms may contribute to this depending on the type of feeding and nature and extent of damage incurred. Mechanisms include damage to vascular tissue and indirect effects on photosynthesis via alterations in leaf hydraulic properties, stomatal aperture, phloem loading and sucrose transport (Nabity et al. 2009). In addition, herbivory injury, or the perception of injury, can induce defence reactions which may include localised cell death from the release of autotoxic defence compounds and a more general down-regulation of photosynthesis related gene expression (Nabity et al. 2009; Tang et al. 2009). It has been suggested that there is a trade-off between photosynthetic metabolism and defence, with a down-regulation of photosynthesis freeing up resources for use in defence reactions (Baldwin 2001; Tang et al. 2009). However, estimates of the carbon requirement for synthesis of defence compounds suggests that the demand is small, and that the down-regulation of photosynthesis is a more general programmed response to biotic stress (Foyer et al. 2007). The negative relationship between defence and photosynthesis is consistent with observations on localised changes in photosynthetic metabolism in incompatible plant pathogen interactions. During race-specific (Mla12) and broad spectrum (mlo) resistance of barley (*Hordeum vulgare*) to powdery mildew (*Blumeria graminis*), photosynthesis was reduced, not only in cells subject to attempted penetration by the fungus, but also in surrounding cells (Swarbrick et al. 2006). The reduction was associated with both localised cell death and altered source-sink relations and carbon metabolism at the sites of resistance. There was also a down-regulation of Rubisco and chlorophyll a/b binding protein genes. These adjustments support the view that there is a fitness "cost" associated with host resistance to pathogens (Smedegaard-Petersen and Tolstrup 1985; Swarbrick et al. 2006).

Modelling canopy photosynthesis of spring barley in response to foliar disease has highlighted the potential impact variation in the effects of infection on photosynthesis in symptomless regions of infected leaves may have on overall disease tolerance (Bingham and Topp 2009). Within the range of parameter values selected, disease tolerance was more sensitive to variation in the virtual lesion size than to changes in canopy architecture and area, suggesting that reducing the virtual lesion size could be a worthwhile target for improving disease tolerance. However, in order to improve disease tolerance through plant breeding, variation in the target traits must exist within the breeding population and breeders must have the means to select for the traits either phenotypically or genetically. To date few studies have quantified within-species variation in responses to pathogen infection that could impact on the size of the virtual lesion. Nevertheless there are indications that some potentially useful intra-specific variation might exist. Zuckerman et al. (1997) reported a 3.5-fold increase in the rate of photosynthesis in remaining green tissue of spring wheat variety Miriam compared to variety Barkai following infection by *M. graminicola*. This was associated with an apparently greater disease tolerance in Miriam and a smaller reduction in mean seed weight. Although winter wheat genotypes infected with rust (*Puccinia recondita*) did not differ in the rate of photosynthesis expressed per unit of remaining green area, the rate of disease-induced leaf senescence did vary which could conceivably influence disease tolerance (Spitters et al. 1990). Recently Ben Slimane et al. (2012) distinguished in wheat leaves inoculated by four strains of *Mycosphaerella graminicola*, (i) the local senescence around inoculation and (ii) the remote apical senescence linked to the natural processes of nitrogen mobilisation during grain filling. Apical senescence was not affected in that case, but no investigation was made on other tissues. More generally, a systematic evaluation of the scale of intra-specific variation in leaf responses to pathogen infection and herbivory is required to determine whether these responses can be modified to improve disease and pest tolerance. This must include a consideration not only of the rate of photosynthesis in the remaining healthy tissue in damaged leaves, but also of the fate of the carbon assimilates produced. Plant fitness will only be sustained if export of assimilates can be maintained from the damaged leaves to support the growth and reproductive yield of the plant as a whole.

Furthermore, the effect of disease on photosynthesis or yield is likely to also depend on environmental or abiotic factors such as light and nitrogen nutrition (Scholes and Rolfe 2009; Robb 2007). Neither Robert et al. (2004) nor Carretero et al. (2011) have found much variation in photosynthetic response of wheat leaves to rust with differing leaf nitrogen. In all cases the estimated values of β were significantly greater than 1.0 indicating that photosynthesis was inhibited beyond the visible lesion, but there was no significant effect of N treatment. Nevertheless Robert et al. (2004) showed that sporulation per unit of sporulating area was decreased in wheat leaves of plants managed at low N levels, suggesting that this aspect of damage may depend on leaf nutrition.

Tolerance at plant level: compensation as a way to tolerance

"Functional" compensation

With functional compensation the reduction in physiological function caused by disease or pests at one location in the plant is compensated for by an increase of the same function at another location. A number of studies have considered adjustments in photosynthetic metabolism in parts of the plant other than the infected or damaged leaves. Increased rates of photosynthesis in healthy leaves of infected plants have been observed in several pathosystems (Ayres 1981; Williams and Ayres 1981; Roberts and Walters 1986; Rooney and Hoad 1989; Murray and Walters 1992). Similar effects have also been reported in response to insect herbivory and mechanical defoliation (Tiffin 2000; Macedo et al. 2006; Nabity et al. 2009). The increases are considered to compensate, at least in part, for the loss of photosynthetic activity in infected or wounded leaves and are often interpreted in terms of responses to changes in source-sink relations (Thomson et al. 2003). The magnitude of the response differs between studies and tends to be larger for dicotyledonous species than monocots (Bingham et al. 2009). It may also depend on the growth stage of the plant. Wheat plants inoculated with *Septoria nodorum* at the three leaves stage showed increased rates of photosynthesis in non-inoculated leaves, whereas a similar treatment imposed when plants had six leaves did not result in an increase (Rooney and Hoad 1989). The mechanisms underlying these differences are not understood. It has been speculated that it may relate to variation in the source-sink balance of the plant at different growth stages and between monocots and dicots (Bingham et al. 2009). Plants whose rate of photosynthesis is restricted by feedback inhibition from a low sink demand for carbon assimilates may be able to up-regulate the rate rapidly in healthy leaves when photosynthetic activity elsewhere is reduced by pathogen infection or herbivory. There may be less scope for making these adjustments if photosynthesis is already operating close to full capacity. Improving tolerance by increasing the ability of plants to compensate through physiological adjustments in healthy leaves may be a more realistic target than seeking reductions in virtual lesion size in infected leaves. Modelling of canopy photosynthesis suggests that disease tolerance of barley is sensitive to this trait (Bingham and Topp 2009). However, as was the case for responses occurring within damaged leaves, at present very little is known about the extent of within-species variation in the capacity for making compensatory adjustments in healthy leaves.

As a general trend, N-uptake is reduced in plants infected either in leaves or in roots. Root diseases have been studied far less intensively than foliar disease, but here too there is some evidence of compensatory adjustments in host physiology. ^{15}N labelling experiments have shown that in wheat plants infected with take-all (*Gaeumannomyces graminis*) N-uptake per unit of root increased, although compensation was not complete and the same level of nutrition as healthy plants was not achieved (Schoeny et al. 2003). However, variation in the extent of this compensation with either the genotype or the environment needs further investigation.

"Morphogenetic" compensation

Over the longer term, compensation in response to biotic stress has often been found to occur through variation in morphogenesis, as is also the case for abiotic stresses. The response can sometimes result in an increase in source capacity because either shoot:root ratio or leaf area ratio increases. Moreover, the new leaves formed under the biotic stress may also show a transient increase in photosynthetic capacity (Ayres 1991), in the same way as adult leaves.

The emergence of new tillers or branches that overcome the carbon loss from disease or herbivory has also

been described as a compensatory response to foliar, stem or spike diseases and pests in natural (Strauss and Aggrawal 1999) or cultivated species (Bastiaans and Roumen 1993; Rosenthal and Welter 1995). It is worthwhile emphasising that the degree of compensation greatly depends on the ability of the crop for morphogenesis; that is to say, its developmental stage at the time the disease or pest attack occurs (Ayres 1991; Bastiaans 1993a). There are only a few documented cases of morphological adjustments in response to root diseases. In wheat infected by take-all, a disease-induced root production was found to be dependant on the genotype, with a marked interaction with the inoculum load (Bailey et al. 2006). In a natural invasive plant species, Newingham et al. (2007) found, for the first time, a rapid nitrogen reallocation from attacked roots to shoots and considered this to be an efficient way to overcome root herbivory and allow better regrowth. New roots with a greater uptake capacity for nitrogen or water have sometimes been found in response to root diseases (Bailey et al. 2005; 2006) and may partly offset the rapid increase shoot: root ratio caused by root parasites (Ayres et al. 1996). This morphological adjustment is in contrast to the increase in shoot: root ratio with foliar diseases (Ayres 1991).

In the case of attacks on reproductive organs by herbivores and pathogens, the loss of floral buds may be compensated in a number of species by the growth of previously set meristems or dormant buds resulting in the emergence of new reproductive sinks (for review see Strauss and Aggrawal 1999; Ayres et al. 1996). The mechanisms involved are associated with broken apical dominance (Sadras 2000) and an alteration in source-sink ratio (Ayres et al. 1996). Their effectiveness in compensating for damage caused by pests or pathogens will depend on whether competent meristems are still available in plants for regrowth and whether they have time to complete development and contribute to yield (Ayres et al. 1996; Tiffin 2002). This capacity has been studied less in cultivated crops than in native vegetation although knowledge of genotypic or environmental variation in the capacity for compensation in cultivated crop species would be of a great use for selection of tolerance of stressful conditions (Pinet 2010). Based on the biology of cultivated species, we may therefore hypothesize that indeterminate crops will have a greater potential for compensating for losses of reproductive organs than determinate ones.

Tolerance may be increased also when the period of crop sensitivity lies outside the period within which the pest is present. In potato (*Solanum tuberosum*) crops, early cultivars are able to partially avoid the negative impacts of disease on yield by completing a greater fraction of the tuber filling period before the epidemic induces premature canopy senescence. Tubers are initiated earlier and leaf growth continues for longer and thus, the period of tuber filling is extended. Therefore, early cultivars suffer less yield loss than late cultivars under similar disease pressure (Neth 1992).

The role of the reserves to buffer the loss of assimilates

Utilization of temporary storage reserves has also been advocated as a way of compensating for losses of carbon or nitrogen in plants attacked by pests and pathogens (Ayres et al. 1996; Gaunt and Wright 1992). However, Ben Slimane et al. (2009), mimicking the effect of disease/herbivory by cutting or masking leaf blades of wheat during grain filling, found no indication for enhancement of N-mobilisation in unaffected organs. By contrast, Serrago et al. (2011) have found that the kinetics of stem soluble carbohydrate (i.e. carbon reserve) utilization in wheat varied with fungicide treatment. Genetic variation in stem soluble carbohydrate reserves has been reported in cereals which could influence the ability of the variety to compensate for reductions in photosynthesis during grain filling (Bingham et al. 2009). Ayres et al. (1996) as well as Parker et al. (2004) also hypothesized that larger plant storage reserves could favour tolerance mostly when disease develops late during grain filling. However Foulkes et al. (2006), comparing numerous genotypes, found a negative correlation between tolerance to Septoria leaf blotch and stem reserves in wheat. This might have occurred because breeding for high-yield has led to short-stem varieties with high numbers of grains together with high stem carbohydrate contents (Parker et al. 2004). Interestingly these results point out that the contribution of stem reserves to tolerance can not be analysed independently from information on the source-sink ratio, and particularly an understanding of how far the availability of assimilate exceeds the assimilate requirement to grow the existing sinks to their potential. The extent and the rate at which compensatory adjustments occur depends largely on the specific interaction the pest and pathogen develops with the host plant. Depending on the relative location and age of healthy

and diseased source leaves or roots, nutrients could either be redirected promptly towards existing organs with high activity or used more slowly to build new source capacities. A reprogramming of primary metabolism is often observed at the cellular scale to achieve these changes, and even if the precise metabolic changes depend on the type of interaction, they often result in changes in source-sink equilibrium and modifications to sugar metabolism and transport (Schwachtje and Baldwin 2008; Walters and McRoberts 2006). As source-sink interplay is highly dependent on resource availability, it is to be expected that the extent to which compensation occurs will also be highly dependent on multiple resource levels (Wise and Abrahamson 2005), although this has not been properly demonstrated as yet. However, it provides an analytical framework to revisit compensatory adjustments in physiology and morphology for crops under fluctuating climatic and management conditions. As both organ nutrition and source-sink interplay are sensitive to plant architecture, the extent of compensation is likely to be in part driven by modifications to plant and crop architecture.

Tolerance at canopy level

For many crops, the effects of aerial disease on yield are consistent if accounted for via the effects on green area index (GAI, m^2 green area m^{-2} ground), accumulated light interception, and the resulting dry matter accumulation and partitioning (Waggoner and Berger 1987; Bryson et al. 1997). In accordance with Beer's law, the proportion of incident radiation intercepted by the canopy is a function of both its size and architecture. A GAI of approximately 6 for wheat is required to intercept 95% of the solar radiation, depending on the extinction coefficient k, which is largely determined by the posture of the leaves within the canopy. The intercepted solar radiation IPAR is related to the GAI by the Beer-Lambert law: IPAR = PARi $(1\text{-}e^{-k\ GAI})$, with PARi as incident radiation. In modern wheat cultivars the distribution of green area results in 80% of the photosynthetic capacity of the crop being attributed to the upper portion of the canopy (Sylvester-Bradley et al. 2012). As leaves do not contribute equally to canopy photosynthesis, because they differ in area and do not receive the same radiation, the vertical distribution of the disease between leaf layers (or injury for herbivory) has a major impact on crop growth for a given architecture. Conversely, for the same vertical distribution of disease or herbivory, variation in canopy architecture may confer relatively more or less tolerance. Bancal et al. (2007) points out that the impact of variation in canopy size depends on the vertical distribution of disease on wheat. For pea infected by *Mycosphaerella pinodes*, Beasse et al. (2000) showed that both combined vertical distributions of leaf area and of disease determine the impact of disease on growth. These authors developed a model that combines the decrease in rate of photosynthesis in the leaves according to the vertical gradient of disease severity and the differences in photosynthetic function of the various layers of the canopy. They showed that the disease spread up the plant from the bottom to the top of the canopy, eventually reaching the layers that contribute significantly to light absorption. Before this particular stage, the impact of disease on growth remained low, but it increased dramatically as disease spread to the upper canopy. Thus, the same necrotic area has a variable effect on crop growth depending on the location of disease within the canopy. In that case, canopy architecture, as governed by vertical leaf area distribution, plays a determinant role in tolerance. Leaf angle and shape may also have an influence. In cereals for instance, large, prostrate upper leaves equate to higher extinction coefficient values because they block penetration of light to the basal leaves. Canopies which have a high extinction coefficient for PAR may confer tolerance if disease is located low in the canopy because a crop with this profile would be less sensitive to loss of leaf tissue to disease or pests low in the canopy (Bingham and Newton 2009). Thickness of leaf also influences the transmission of radiation within the canopy (Kramer et al. 1980) and will affect the extinction coefficient value.

For oilseed rape (*Brassica napus*), as in many crop species, yield is largely driven by the SN, which is determined by the survival of branches, flowers and young pods (Diepenbrock 2000). Pod survival is related to the amount of intercepted PAR, during the flowering period (Leterme 1988). In this planophyll crop, a GAI of 3–4 is sufficient to intercept the required amount of solar radiation. Canopies which are larger may paradoxically reduce the amount of intercepted light reaching green leaf and stem tissue because the dense flower layer reflects a proportion of light away from the canopy, and thus reduces the number of seeds set per pod. Therefore, damage to flowers caused by pests such as the pollen beetle generally has little impact on yield at the beginning

of flowering because light is reaching leaves and branching can compensate losses. As the number of seeds that are growing increases with time, less assimilate is available to new branches or flowers. Thus the compensation capacity of the plant diminishes during the reproductive period. Yield losses depend on both the intensity and the time of the attack. More determinate varieties (or higher sowing density) are theoretically less tolerant of beetles, because of their poorer compensation capacity.

In the context of west European agriculture, several major genetic changes in wheat plants over the last three decades have led to visible changes in canopy architecture, brought about by phenotypic selection. Flag leaves have become significantly smaller and culm leaves have become more erect. Foulkes et al. (2006) showed that a large flag leaf size was associated with greater disease tolerance if the spread of foliar disease to the flag leaf could be avoided because, for foliar diseases of wheat, the dominant effect appears to be a reduction in post-anthesis radiation interception (Robert et al. 2004).

Quantification of tolerance

Techniques to measure tolerance

This section considers the challenges associated with measuring tolerance, in order to identify tolerant germplasm and associate particular physiological traits with tolerance. The key issues, their implications and potential methods to address them are considered below in reference to fungal disease. Similar considerations must also be taken into account when quantifying tolerance to insect pests.

Tolerance can be measured in both controlled environment and field experiments depending on the objectives of the study. Controlled environment conditions would be especially useful to establish knowledge or test hypotheses concerning the mechanisms of tolerance and the potential contribution of selected traits, for instance a reduction in the virtual lesion size or increased capacity for compensation. Yet tolerance is likely to be the sum of multiple processes, which interact highly depending on the growth conditions; interactions and conditions that will be difficult to reproduce in controlled conditions. Many of the potential tolerance mechanisms discussed above operate at the canopy/crop scale, rather than at the level of the individual plant or organ. Hence, tolerance will usually need to be quantified in field experiments, rather than in controlled environments. In controlled environments and to even greater extent in field experiments, it is almost impossible to obtain similar levels of disease severity across a range of contrasting cultivars or breeding lines, to allow a direct comparison of tolerance. Hence, it is necessary to quantify tolerance as the rate of change of yield (or other measure of growth) per unit disease severity (or a surrogate for severity). This requires a range of severities to be obtained on each cultivar or line, within an experiment. This could be obtained using various levels of inoculation in controlled environment conditions, but in field trials, it is most reliably obtained by using contrasting doses of fungicide treatment. In the field it is difficult to generate the required gradient of disease severity via inoculation because many factors influence the development of disease. Realistically, it is often only possible to set up ‘high disease’ and ‘low disease’ treatments. Low disease may come from the background level at a relatively low disease-risk site and high disease from additional inoculation. There is greater flexibility for setting up a gradient of severity when the pathogen can be applied as a spore suspension, but this often requires large quantities of inoculum and frequent applications and is, therefore, expensive and time-consuming. A much greater contrast in severity can usually be achieved reliably by growing a crop at a high disease risk site and then controlling the epidemic by the application of fungicides.

Avoiding bias with fungicide use

The use of fungicides, however, raises the issue of whether the fungicide treatment used might bias the results, through direct effects of the active substance on the physiology of the crop. For example in some barley genotypes there is evidence of significant yield responses to fungicide application in the relative absence of visible disease (Bingham et al. 2012). The use of non-systemic protectant fungicides largely overcomes the concern about direct physiological effects, but may not provide sufficiently effective disease control. Where the use of systemic fungicides is required, the use of quinone outside inhibitors (QoI) and “new generation” succinate dehydrogenase inhibitors (SDHI)

with known physiological effects should be avoided. Some possibility of bias remains, but may not be of practical concern because the fungicide requirement of a cultivar is primarily driven by the size of the difference between fungicide treated and untreated yields (Paveley et al. 2008). Hence, if selection of breeding material is based on measures of tolerance from comparisons of treated/untreated yields, then the practical aim (reduced fungicide dependence) will be achieved.

Minimising genotype and environment interactions

Environmental variation can result in substantial genotype by environment interactions. If these interactions are of too large magnitude, this may indicate that the particular form of tolerance being tested may be too unreliable to be of overall value, except perhaps within genotypes adapted to a narrow range of environments. However, if the interactions are of intermediate scale, the heritable variation within a set of germplasm may be sufficiently large to be of value, but difficult to identify through the "noise" created by the interactions with environment.

Measurement of tolerance using percentage severity values (or integrals thereof) as the independent variable, is particularly prone to environmental variation, due to site and seasonal variation in canopy size. As Waggoner and Berger (1987) pointed out: "…neither the rate of change of x [dimensionless severity] nor the integral of x tell the size of the foliar factory, how long it operates or how much insolation it absorbs." Quantifying tolerance using a surrogate for severity which is more closely related to resource capture (e.g. post-anthesis healthy area duration for wheat), reduces the environmental "noise" but precludes detection of some tolerance traits (e.g. if healthy area duration is used, tolerance operating by reduced green area loss per unit symptom severity would not be detected). Hence, the choice of the variable used to quantify tolerance is an important decision, which is discussed further by Bingham et al. (2009).

Efficient use of experimental resources

Because the range of variation found in a trait is related to the size of the population measured, it may be necessary to quantify tolerance in large numbers of cultivars or breeding lines in order to identify lines which express tolerance to a high degree. Also, if the aim is to enable genotypic selection, by identifying QTL associated with tolerance (or the sub-traits which determine tolerance), then more than 100 lines will need to be phenotyped to enable QTL analysis. However, precise and accurate quantification of tolerance is resource intensive, which limits the number of cultivars/lines which can be screened.

A simple estimation of tolerance is obtained by regression analysis as the slope of a relationship of yield on disease severity (or a surrogate for severity); but the relationship of disease to yield may be not linear, and thus needing more coefficients that a single slope. Nevertheless the following rationales still apply. The validity of the regression and the confidence intervals on the coefficient will depend to a large extent on the distribution and range of variation along the independent variable. A large range of variation can be obtained by fungicide treated/untreated contrasts, but unless the variation between replicate values is substantial, the independent variable will tend to be clustered at either end of the regression, making the analysis less valid. Nevertheless, this approach has been used successfully (Parker et al. 2004; Foulkes et al. 2006). Including intermediate fungicide doses improves the validity of the analysis and the confidence in the slope coefficient, but increases the number of plots required to test each cultivar or line. Similarly, estimates of the slope coefficient will be increased by taking more sequential assessments of the severity measure during the yield-forming period, to improve the calculation of the integral (for example of healthy area duration). However, the resources required for assessments increase in proportion to the number of measurement dates, thus decreasing the germplasm which can be assessed. Automation may ultimately resolve this issue, but current methods for assessing GAI by light interception or reflectance are insensitive at GAI values above approximately five. Most crop species have GAI values higher than this during the yield forming period, in order to intercept most of the available incident radiation. Finally, where large numbers of treatments or genotypes are tested in field experiments, consideration needs to be given to accounting for spatial variation by appropriate experimental designs, such as the inclusion of multiple reference cultivars within each replicate block, and accounting for gradients in variables across the trial area during analysis of variance.

One approach to reconcile the need to screen large numbers of lines and the need for intensive

measurements could be to: (i) start by screening a wide range of germplasm, using minimal treatments and assessments, e.g. in controlled conditions; (ii) identify a small number of cultivars/mapping population lines which contrast most strongly for tolerance; (iii) use detailed physiological measurements on the contrasting lines to identify key sub-traits associated tolerance; (iv) phenotype the key traits in experiments on 100+ mapping population lines.

Models of diseased and pest-damaged crops

How modelling is useful to quantify damage and prioritize tolerance traits

Since correlations between traits on the one hand and tolerance on the other hand in experimental data sets may be due to mechanistic links or autocorrelation (e.g. due to genetic linkage), more rapid progress could be made by a combination of experimentation and mathematical modelling, to identify beneficial trait combinations. Modelling appears to be a useful approach to quantify tolerance and compare varieties, and to virtually define ideotypes in different environmental conditions.

Overall, attempts to simulate tolerance have focused mainly on carbon-based growth modelling, thus neglecting multiple stress interactions, either between different biotic stresses or between biotic and abiotic stresses. As the impact of multiple stresses has long been shown to be non-additive (Johnson 1992; see review of Atkinson and Urwin 2012), evaluating tolerance by using models under a large range of conditions is required to avoid under- or over- estimating losses, and thus the contribution of particular traits to tolerance. Several attempts to use models to investigate tolerance traits have been made (Beasse et al. 2000; Parker et al. 2004; Bingham and Topp 2009; Carretero et al. 2010). The growth model of Beasse et al. (2000) for pea infected by *Mycosphaerella pinodes* previously described has been used to compare six cultivars with contrasting vertical distribution of leaf area (Le May et al. 2005). Growth and yield were found to be very different among varieties and years despite no significant differences for the relationship between disease severity and photosynthesis (i.e. virtual lesion size) at the leaf scale. Growth was well simulated by the Beasse's model for all genotypes and years showing that the differences were only due to the interplay between vertical distribution of leaf area and of disease. These results suggest that for the same vertical distribution of disease, taller cultivars should be more tolerant *per se*. It would have been interesting to couple this with the relation between vertical epidemic distribution and canopy structure to optimise the architecture (Le May et al. 2009).

Using a growth model, Bingham and Topp (2009) performed a sensitivity analysis to hierarchize the traits most involved in disease tolerance of barley. Interestingly, little variation in tolerance was predicted with disease severity, which suggests that traits identified in one situation will still apply elsewhere. The capacity to increase the photosynthetic potential (P_{max}) of healthy leaves in response to disease elsewhere on the plant and the size of the virtual lesion (inactivated area around disease symptoms, as defined in Bastiaans, (1993b)) in infected leaves had the greatest impact on tolerance. The size of the virtual lesion is well known to vary among pathosystems; unfortunately, there is little evidence in the literature for such variation under different environments within a given pathosystem (see previous sections). On the other hand, the interaction of virtual lesion size with disease distribution (uniform, intermediate or concentrated on the lower leaf layers) impacted on tolerance, because of the non-linear relationship between photosynthesis and incoming radiation. The direct effect of canopy architecture on tolerance was balanced: high values of both GAI and the extinction coefficient k might increase disease tolerance, but might also lead to a decline in yield potential or excessive maintenance respiration in the absence of disease. Indeed the strong impact on tolerance of disease distribution may result from architecture variations. Furthermore, erectophyll canopies combined with top-distributed diseases, as well as planophyll canopies combined with bottom-distributed diseases were identified as the best way to insure increased tolerance (Bingham and Topp 2009; Carretero et al. 2010).

Many of the preceding models however did not consider whole plant nutrition even though as noted above the source-sink balance has often been shown to interfere with tolerance. Others have used constant partitioning coefficients for every sink, thus discarding by the way the compensatory potential of reserves. Recently Bancal et al. (2012) quantified the fungal sink competitiveness of *Puccinia triticina* with wheat grain filling. In this case where a biotrophic foliar

pathogen does not invade vessels, the pathogen was regarded as an additional sink transplanted into a plant. The authors thus enhanced an existing source-sink model (Bancal and Soltani 2002) with two sources (photosynthesis and reserve hydrolysis) and two sinks (grain growth and reserve polymerization) by adding the fungal sink (spore production). This study pointed out that not only photosynthesis, but also sink activities and compensation for reductions in photosynthesis by the reserve pool depended non-linearly on the incoming radiation level. It suggests that disease nutrition effects and tolerance are both modulated by climate. In the case of tree-pest interactions or for pathogens invading vessels, models accounting for resistances to carbon fluxes should be used as proposed by Minchin and Lacointe (2005). They could simulate for example, the strong interactions that were experimentally found between tree architecture and sink strength in galling efficiency of cottonwood aphids (Larson and Whitham 1997). Lastly, even the effect of necrotrophic pathogens or herbivores, which decrease source capacity, but could not be considered as additional sinks to plants, might be accounted for by such a modelling framework because of the non-linear interplay between the reduced sources and existing plant sinks. Interestingly, growth and repartition processes are affected by climatic conditions; therefore any given crop suffering the same epidemic will develop differential damage depending on the weather conditions. Combining long-term climatic records with simple wheat canopy models during grain filling, Bancal et al. (2010) suggested that up to 50% of the variation in damage caused by late foliar pathogens is linked to weather. This will complicate any climate-free quantification of tolerance, e.g. for the genetic improvement of tolerance. Conversely, one could imagine a combination of cultivar choice and crop management designed to escape the most sensitive period in a given climatic area to maximize tolerance.

Short and long term reallocations of carbon have long been highlighted as a key process in tolerance to foliar or root herbivory. Models designed for evolutionary goals, may well be adapted to crops and could open the way to a better understanding of tolerance traits, providing they can be further linked to resource-based crop models. Modifications of shoot:root ratios are often mentioned to predict plant compensation of damage caused by foliar or root pests and pathogens (Matyssek et al. 2005; Schwachtje et al. 2006; Newingham et al. 2007). The availability of dormant buds set by architecture also plays a key role in tolerance to herbivory Strauss and Aggrawal (1999), Jullien et al. (2010) and Lehtilä (2000) modelled compensatory regrowth by gradual activation of dormant buds following single or repeated injury events. Seed production may compensate, or even over-compensate, for damage depending on both the timing and the intensity of herbivory and on plant phenology. In the case of mutualism with mycorhizal fungi, that develop relationships with plants that may derive from tolerance strategies (Oliver et al. 2009), Vannette and Hunter (2011) described the transition between mutualism and parasitism as non-linearly related to nutrient limitations. Gayler et al. (2008) developed a dynamic model of carbon and nitrogen repartition between growth and defence compounds. An important output of their study was that growth has the priority over defence in carbon allocation, but, is more sensitive to nitrogen depletion, resulting in varying defence allocation patterns depending on age and environment. Finally, Atkinson and Urwin (2012) advisedly point out examples where selecting for tolerance to one stress resulted in increased or decreased susceptibility to other stresses, thus highlighting the need for modelling tolerance to multiple stresses.

How modelling is useful to improve crop tolerance

Pursuing the objective of improving crop tolerance through the large diversity of crop functions discussed above demands that two practical questions be answered. First of all, what crop functions will provide the highest and/or most consistent gain in tolerance? And secondly, what traits need to be observed to properly select appropriate genotypes or cropping practices? This can be exemplified by work carried out by crop physiologists and breeders concerning adaptation to drought stress, which we will briefly illustrate to introduce the equivalent approach that can be carried out for tolerance to wheat foliar diseases. Starting with a conceptual model of desirable crop functions in a given stress context (Reynolds et al. 2007), the two questions asked above were answered as follows. First of all, both empirical data (Condon et al. 2004, Fig 3) and modelling work (Condon et al. 2004, Fig 5) allowed for key traits (namely transpiration) to be targeted to those environments where they will provide the appropriate response. Secondly, phenotyping for the desired crop functions was adapted to the different constraints

imposed by experimentation at different stages of research and breeding programmes (Reynolds et al. 2009). In the case of wheat foliar diseases, a simple conceptual approach of tolerance is illustrated Fig. 2: it extends the 'Steps model' proposed by Paveley et al. (2001) to link fungicide dose to yield. This model design might still be improved to describe the effects of diseases affecting sink as well as source and thereby enlarge its applicability to different situations. According to Fig. 2, at each step, genotypic variations around either a mean response curve or a curve from a reference genotype should offer a way to quantify components of tolerance. Mechanisms concerning steps 3 to 5, from green area to intercepted radiation (IPAR) to crop growth and finally to yield have been discussed extensively above. The first two steps are relatively less well characterized, but existing relations between fungal biomass and symptoms (Fraaije et al. 2001; Gouache et al. 2011) and symptom area and green area (Robert et al. 2004) have been reported. The 'Steps model' can also be used to integrate the different tolerance mechanisms making it possible to properly deploy them. This is illustrated with steps 3 to 5. As has been stated many times above, yield loss can be linked to symptoms through effects on reducing the life time of green area, which can be accounted for using the inflexion point of GAI (Gooding et al. 2000) or the integral of green LAI during grain filling (usually designed as LAD (leaf area duration), Bancal et al. 2009). Integrating over steps 3 to 5, it is possible to compare varieties that exhibit different LAD to yield relations (Gate et al. 2006). As the "Steps model" illustrates however, the first step for yield building, linking GAI to radiation interception, is curvilinear. Hence, it is conceptually simple to state that the slope of GAI to yield is greater at low GAI. The consequence is that tolerance will be greater at high GAI (or LAD) values than at low values of disease-free crops. Ranking of tolerance between cultivars may thus vary as a function of the magnitude of LAD. Figure 3 indicates how genotypes may show a varying level of tolerance, depending on the LAD that can be reached in a specific environment. Obviously the genotypes exhibit the same tolerance level once the plateau value for LAD is reached, regardless of differences in both LAD and yield potential among them. Yet there is no reason that every genotype, (i) reaches the yield plateau at the same LAD level and (ii) declines its yield according to LAD following the same slope. In the example given, the genotype 1 needs a greater LAD than genotype 2 to reach its yield potential. Thus, at intermediate LAD, genotype 2

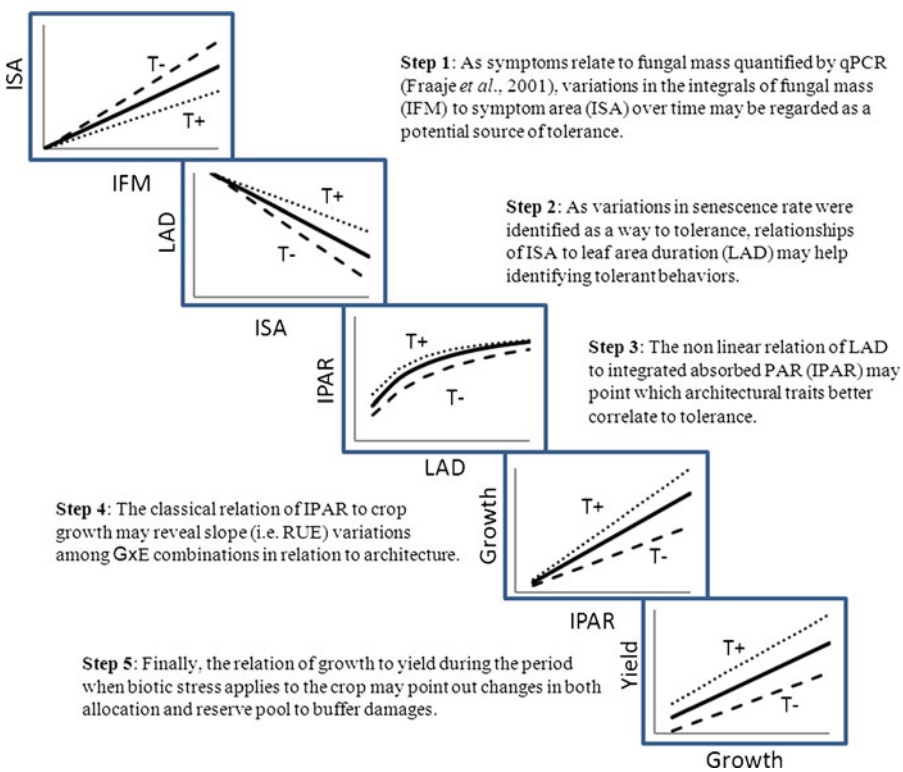

Fig. 2 Different steps to account for tolerance components modified from Paveley et al. (2001). Yield in a diseased plant is achieved through five successive steps, each of them exhibiting tolerant or intolerant behaviors

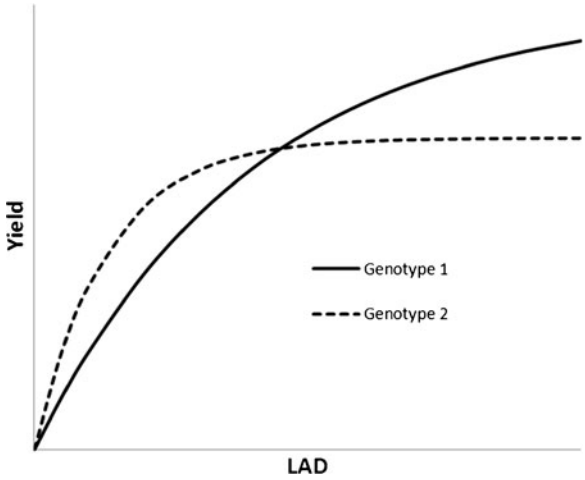

Fig. 3 Yield as a function of LAD after ear emergence for 2 hypothetical cereal genotypes: tolerance is greater when the slope of the LAD-yield is flatter, thus tolerance depends on the range of attainable LAD in various environments

exhibits a lower slope, i.e. a higher tolerance (and in the example a higher yield), whereas genotype 1 shows the highest yield potential at high LAD. Variations in the slope may represent a simple way to characterize tolerance of genotypes to biotic stresses at a given level of LAD.

When evaluating the impact on tolerance of observed genotypic variability at a given step, the 'Step model' can thus account for the expected GAI/LAD range in non-diseased crops, and allow the impact of the trait to be properly evaluated. This may provide a first approach to rapidly screen cultivars. However, as it integrates many steps, a limit of the approach may be to miss out on underlying genotypic variation at one step that is compensated at another. Another interesting point in using the overall LAD to yield response of cultivars is that the effects of other stresses, such as nitrogen or drought, on yield are also mediated (at least partly) through GAI. Consequently, tolerance mechanisms identified in this manner may be of wider use than sole tolerance to foliar pathogens.

Selecting traits as targets for improving tolerance also needs to account for correlations between traits. For example, Arraiano et al. (2009) showed that leaf length, canopy height and leaf prostrateness exhibited correlations in a set of 226 cultivars and breeding lines. Modelling may help to characterize the effects of individual traits, all other parameters being equal, and thus aid in prioritizing targets. Empirical data and modelling are in this case highly complementary. Finally, targeted traits may also have a strong influence on other crop functions: for example, traits influencing canopy light interception discussed above are also targeted to improve yields of healthy crops (Long et al. 2006; Parry et al. 2011), and may also influence disease progress (Lovell et al. 1997). Again, modelling may be used to analyze trait modifications on multiple criteria (Robert et al. 2010) and analyze potential trade-offs or synergies.

Conclusion

Tolerance is a particularly desirable characteristic when preventive measures against disease and pests are inaccessible or inefficient, which is increasingly the case. European pesticide legislation is continuing to reduce the possibilities of using fungicides and insecticides. Host plant resistance, although a key solution, is not as adaptable as the use of pesticides: once a variety is sown, the pest populations and disease epidemics to which the crop will be sensitive is set. For example, when choosing a disease resistant variety, the most frequently damaging disease is generally targeted. However, this may not be the actual disease(s) that develops during the growing season. Pathogen population shifts may be responsible (Goyeau et al. 2011; de Vallavieille-Pope et al. 2012), as well as climate variability (for instance the dramatic and unexpected brown rust epidemic in northwest Europe in 2007 as reported by West et al. 2012). Thus, tolerance provides a useful and complementary approach to resistance, especially to deal with the variability inherent to disease epidemics and pest populations. This may be all the more important under a changing climate (Shaw and Osborne 2011). Moreover, Espinosa and Fornoni (2006) have also shown that host tolerance has no effect on the performance of enemies, and could thus avoid coadaptation of pests to hosts; however these authors also pointed out that the expression of tolerance may depend either on the inoculum load or the genotype × environment.

Simulation models that attempt to predict the effects of damage on crop growth provide a powerful tool to evaluate potential tolerance traits. Advances in the understanding of plant pest and pathogen interactions are required to combine the main processes involved in tolerance expression.

Acknowledgments Work on tolerance carried out by MO Bancal, P Bancal and D Gouache is supported by the French Ministry of Agriculture through a "Contrat de Branche" grant. IJ Bingham thanks Dale Walters (SRUC) for valuable discussions. SRUC receives financial support from the Scottish Government's Rural Environment Science & Analytical Services Division (RESAS).

References

Aguado, A., De Los Santos, B., Gamane, D., Garcıá del Moral, L. F., & Romero, F. (2010). Gene effects for cotton-fiber traits in cotton plant (*Gossypium hirsutum* L.) under Verticillium conditions. *Field Crops Research, 116*, 209–217.

Arraiano, L. S., Balaamb, N., Fenwickc, P. M., Chapman, C., Feuerhelm, D., Howell, P., et al. (2009). Contributions of disease resistance and escape to the control of *Septoria tritici* blotch of wheat. *Plant Pathology, 58*, 910–922.

Atkinson, N. J., & Urwin, P. E. (2012). The interaction of plant biotic and abiotic stresses: from genes to the field. *Journal of Experimental Botany Darwin Review*. doi:10.1093/jxb/ers100.

Ayres, P. G. (1981). Powdery mildew stimulates photosynthesis in uninfected leaves of pea plants. *Phytopathologische Zeitschrift, 100*, 312–318.

Ayres, P G. (1991). Growth responses induced by pathogens and other stresses. In Mooney, H.A., Winner, W.E. & Pell E.J. (Eds.), *Response of plants to multiple stresses* (pp. 227–248). Academic Press Inc.

Ayres, P. G., Press, C. M., & Spencer-Phillips, P. T. N. (1996). Effects of Pathogens and parasitic plants on source-sink relationships. In E. Zamski & A. A. Schaffer (Eds.), *Photoassimilate distribution in plants and crops: Source-sink relationships* (pp. 479–499). New York: M. Dekker Inc.

Bailey, D. J., Paveley, N., Pillinger, C., Foulkes, M. J., Spink, J., & Gilligan, C. A. (2005). Epidemiology and chemical control of take-all on seminal and adventitious roots of wheat. *Phytopathology, 95*, 62–68.

Bailey, D. J., Kleczkowski, A., & Gilligan, C. A. (2006). An epidemiological analysis of the role of diseased-induced root growth in differential response of two cultivars of winter wheat to infection by *Gaeumannomyces graminis* var. *tritici*. *Phytopathology, 96*, 510–516.

Baldwin, I. T. (2001). An ecologically motivated analysis of plant-herbivore interactions in native tobacco. *Plant Physiology, 127*, 1449–1458.

Bancal, P., & Soltani, F. (2002). Source-sink partitioning. Do we need Munch? *Journal of Experimental Botany, 53*, 1919–1928.

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

Bancal, M.-O., Bancal, P., Ben Slimane, R., Gate, P., & Gouache, D. (2009). *Tolérance du Blé à la septoriose. In 9ème Conférence Internationale sur les Maladies des Plantes* (pp. 339–346). Tours: AFPP.

Bancal, P., Gouache, D., Soulard, T. & Bancal, M-O. (2010). A general cultivar tolerance to stresses and its modeling in the case of wheat affected by STB (*Septoria tritici*). In Wery, J., Shili-Touzi, I. & Perrin, A. (Eds.), *Proceedings of Agro2010 - the XIth ESA Congress* (pp. 101–102). Agropolis International Editions, Montpellier, France.

Bancal, M.-O., Hansart, A., Sache, I., & Bancal, P. (2012). Modeling fungal sink competitiveness with grains for assimilates in wheat infected by a biotrophic pathogen. *Annals of Botany, 110*, 113–123.

Bastiaans, L. (1991). The ratio between virtual and visual lesion size as a measure to describe reduction in leaf photosynthesis of rice due to leaf blast. *Phytopathology, 81*, 611–615.

Bastiaans, L. (1993a). Effects of leaf blast on growth and production of a rice crop. 1. Determining the mechanism of yield reduction. *Netherlands Journal of Plant Pathology, 99*, 323–334.

Bastiaans, L. (1993b). Effects of leaf blast on photosynthesis of rice. 1. Leaf photosynthesis. *European Journal of Plant Pathology, 99*, 197–203.

Bastiaans, L., & Kropff, M. J. (1993). Effects of leaf blast on photosynthesis of rice. 2. Canopy photosynthesis. *Netherlands Journal of Plant Pathology, 99*, 205–217.

Bastiaans, L., & Roumen, E. C. (1993). Effect on leaf photosynthetic rate by leaf blast for rice cultivars with different types and levels of resistance. *Euphytica, 66*, 81–87.

Beasse, C., Ney, B., & Tivoli, B. (2000). A simple model of pea (*Pisum sativum* L.) growth affected by *Mycosphaerella pinodes*. *Plant Pathology, 49*, 187–200.

Beattie, G., & Lindow, S. E. (1995). The secret life of foliar bacterial pathogens on leaves. *Annual Review of Phytopathology, 33*, 145–172.

Ben Slimane, R., Bancal, P., & Bancal, M.-O. (2009). Regulation of grain N accumulation in wheat. In F. Feldman, D. V. Alford, & C. Furk (Eds.), *Crop plant resistance to biotic and abiotic factors (pp 198–208)*. Braunschweig: Deutsche Phytomedezinische Gesellschaft.

Ben Slimane, R., Bancal, P., Suffert, F., & Bancal, M.-O. (2012). Localized septoria leaf blotch lesions in winter wheat flag leaf do not accelerate apical senescence during the necrotrophic stage. *Journal of Plant Pathology, 1*. doi:10.4454/JPP.FA.2012.055.

Berger, S., Papadopoulos, M., Schreiber, U., Kaiser, W., & Roitsch, T. (2004). Complex regulation of gene expression, photosynthesis and sugar levels by pathogen infection in tomato. *Physiologia Plantarum, 122*, 419–428.

Bingham, I. J., & Newton, A. C. (2009). Crop tolerance of foliar pathogens: possible mechanisms and potential for exploitation. In D. R. Walters (Ed.), *Disease Control in Crops. Biological and Environmentally Friendly Approaches* (pp. 142–161). Oxford: Wiley-Blackwell.

Bingham, I. J., & Topp, C. F. E. (2009). Potential contribution of selected canopy traits to the tolerance of foliar disease by spring barley. *Plant Pathology, 58*, 1010–1020.

Bingham, I. J., Blake, J., Foulkes, M. J., & Spink, J. (2007a). Is barley yield in the UK sink limited? I. Post-anthesis radiation interception, radiation use efficiency and source-sink balance. *Field Crops Research, 101*, 198–211.

Bingham, I. J., Blake, J., Foulkes, M. J., & Spink, J. (2007b). Is barley yield in the UK sink limited? II. Factors affecting potential grain size. *Field Crops Research, 101*, 212–220.

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

Bingham, I.J., Hoad, S.P, Thomas W.T.B. & Newton A.C. (2012). Yield response to fungicide of spring barley genotypes differing in disease susceptibility and canopy structure. Field Crops Research (in press).

Bryson, R. J., Paveley, N. D., Clark, W. S., Sylvester-Bradley, R., & Scott, R. K. (1997). Use of in-field measurements of green leaf area and incident radiation to estimate the effects of yellow rust epidemics on the yield of winter wheat. *European Journal of Agronomy, 7*, 53–62.

Caldwell, R. M., Schafer, J. F., Compton, L. E., & Patterson, F. L. (1958). Tolerance to cereal leaf rusts. *Science, 128*, 714–715.

Carretero, R., Serrago, R., Bancal, M.-O., Perello, A. E., & Miralles, D. J. (2010). Absorbed radiation and radiation use efficiency as affected by foliar diseases in relation to their vertical position into the canopy in wheat. *Field Crops Research, 116*, 184–195.

Carretero, R., Bancal, M.-O., & Miralles, D. J. (2011). Effect of leaf rust (*Puccinia triticina*) on photosynthesis and related processes of leaves in wheat crops grown at two contrasting sites and with different nitrogen levels. *European Journal of Agronomy, 35*, 237–246.

Chou, H. M., Bundock, N., Rolfe, S. A., & Scholes, J. D. (2000). Infection of *Arabidopsis thaliana* leaves with *Albugo candida* (white blister rust) causes a reprogramming of host metabolism. *Molecular Plant Pathology, 1*, 99–113.

Clarke, D. D. (1984). Tolerance of parasitic infection in plants. In R. K. S. Wood & G. J. Jellis (Eds.), *Plant diseases: infection damage and loss*. Oxford, UK: Blackwell Scientific.

Clarke, D. D. (1986). Tolerance of parasites and disease in plants and its significance in host-parasite interactions. *Advances in Plant Pathology, 5*, 161–197.

Condon, A., Richards, R., Rebetzke, G., & Farquhar, G. (2004). Breeding for high water-use efficiency. *Journal of Experimental Botany, 55*, 2447–2460.

Cooper, J. I., & Jones, A. T. (1983). Responses of plants to viruses: proposals for the use of terms. *Phytopathology, 73*, 127–128.

De Vallavieille-Pope, C., Ali, S., Leconte, M., Enjalbert, J., Delos, M., & Rouzet, J. (2012). Virulence dynamics and regional structuring of *Puccinia striiformis* f. sp. *tritici* in France between 1984 and 2009. *Plant Disease, 96*, 131–140.

Diepenbrock, W. (2000). Yield analysis of winter oilseed rape (*Brassica napus* L.): a review. *Field Crops Research, 67*, 35–49.

Espinosa, E. G., & Fornoni, J. (2006). Host tolerance does not impose selection on natural enemies. *New Phytologist, 170*, 609–614.

Fischer, R. A., & Stockman, Y. M. (1980). Kernel number per spike in wheat (*Triticum aestivum* L.): Responses to preanthesis shading. *Australian Journal of Plant Physiology, 7*, 169–180.

Fornoni, J., Nunez-Farfan, J., Luis Valverde, P., & Rauscher, M. D. (2004). Evolution of mixed strategies of plant defense allocation against natural enemies. *Evolution, 58*, 1685–1995.

Foulkes, M. J., Paveley, N. D., Worland, A., Welham, S. J., Thomas, J., & Snape, J. W. (2006). Major genetic changes in wheat with potential to affect disease tolerance. *Phytopathology, 96*, 680–688.

Foyer, C. H., Noctor, G., & van Emden, H. F. (2007). An evaluation of the costs of making specific secondary metabolites: does that yield penalty incurred by host plant resistance to insects result from competition for resources? *International Journal of Pest Management, 53*, 175–182.

Fraaije, B. A., Lovell, D. J., Coelho, J. M., Baldwin, S., & Hollomon, D. W. (2001). PCR-based assays to assess wheat varietal resistance to blotch (*Septoria tritici* and *Stagonospora nodorum*) and rust (*Puccinia striiformis* and *Puccinia recondita*) diseases. *European Journal of Plant Pathology, 107*, 905–917.

Gallagher, J. N., Briscoe, P. V., & Hunter, B. (1975). Effects of drought on grain growth. *Nature, 264*, 541–542.

Gambín, B. L., Borrás, L., & Otegui, M. E. (2006). Source-sink relations and kernel weight differences in maize temperate hybrids. *Field Crops Research, 95*, 316–326.

Gao, H., Beckman, C. H., & Mueller, W. C. (1995). The nature of tolerance to *Fusarium oxysporum* f. sp. *lycopersici* in polygenically field-resistant marglobe tomato plants. *Physiological and Molecular Plant Pathology, 46*, 401–412.

Garry, G., Jeuffroy, M.-H., Ney, B., & Tivoli, B. (1998). Photosynthesis of leaves of pea (*Pisum sativum* L.) infected by *Mycosphaerella pinodes*. *Plant Pathology, 47*, 473–479.

Gate, P., Gouache, D., Robert, C., Bancal, M.-O., Lannou, C., & Ney, B. (2006). *What plant variable to account for loss due to septoria leaf blotch in winter wheat? In 8ème Conférence Internationale sur les Maladies des Plantes* (pp. 348–358). Tours, France: AFPP.

Gaunt, R. E., & Wright, A. C. (1992). Disease yield relationships in barley. II. Continuation of stored stem reserves to grain filling. *Plant Pathology, 41*, 688–701.

Gayler, S., Grams, T. E. E., Heller, W., Treutter, D., & Priesack, E. (2008). A dynamical model of environmental effects on allocation to carbon-based secondary compounds in juvenile trees. *Annals of Botany, 101*, 1089–1098.

Godin, C., Costes, E., & Sinoquet, H. (1999). A method for describing plant architecture which integrates topology and geometry. *Annals of Botany, 84*, 343–357.

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

Gouache, R., Thepot, S., Roisin-Fichter, C., & Selim, S. (2011). Exploring quantitative relationships between mycelial growth and visual symptoms of *Septoria tritici*. In E. Duveiller & P. K. Singh (Eds.), *8th International Symposium on Mycosphaerella and Stagonospora diseases of cereals* (p. 77). Mexico City: CIMMYT.

Goyeau, H., Berder, J., Czerepak, C., Gautier, A., Lanen, C., & Lannou, C. (2011). Low diversity and fast evolution in the population of *Puccinia triticina* causing durum wheat leaf rust in France from 1999 to 2009, as revealed by an adapted differential set. *Plant Pathology, 61*, 761–772.

Johnson, K. B. (1992). Evaluation of a mechanistic model that describes potato crop losses caused by multiple pests. *Phytopathology, 82*, 363–369.

Jullien, A., Mathieu, A., Allirand, J.-M., Pinet, A., de Reffye, P., Cournede, P.-H., et al. (2010). Characterization of the

interactions between architecture and source-sink relationships in winter oilseed rape (*Brassica napus*) using the GreenLab model. *Annals of Botany, 107*, 765–779.

Kluth, C., & Varrelmann, M. (2010). Maize genotype susceptibility to *Rhizoctonia solani* and its effect on sugar beet crop rotations. *Crop Protection, 29*, 230–238.

Kover, P. X., & Schaal, B. (2002). Genetic variation for disease resistance and tolerance among Arabidopsis thaliana accessions. *Proceedings of the National Academy of Sciences of the United States of America, 99*, 11270–11274.

Kramer, T., Gildemacher, B. H., van der Ster, M., & Parlevliet, J. E. (1980). Tolerance of spring barley cultivars to leaf rust. *Euphytica, 29*, 209–216.

Larson, K. C., & Whitham, T. G. (1997). Competition between gall aphids and natural plant sinks: plant architecture affects resistance to galling. *Oecologia, 109*, 575–582.

Le May, C., Schoeny, A., Tivoli, B., & Ney, B. (2005). Improvement and validation of a pea crop growth model to simulate the growth of cultivars infected with Ascochyta blight (*Mycosphaerella pinodes*). *European Journal of Plant Pathology, 112*, 1–12.

Le May, C., Ney, B., Lemarchand, E., Schoeny, A., & Tivoli, B. (2009). Effect of pea plant architecture on spatiotemporal epidemic development of ascochyta blight (*Mycosphaerella pinodes*) in the field. *Plant Pathology, 58*, 332–343.

Lehtilä, K. (2000). Modelling compensatory regrowth with bud dormancy and gradual activation of buds. *Evolutionary Ecology, 14*, 315–330.

Leterme, P. (1988). *Modélisation du fonctionnement du peuplement de colza d'hiver en fin de cycle: Elaboration des composantes finales du rendement. In Colza: Physiologie et Elaboration du Rendement* (pp. 124–129). Paris: INRA/CETIOM.

Long, S. P., Zhu, X.-G., Naidu, S. L., & Ort, D. R. (2006). Can improvement in photosynthesis increase crop yields? *Plant, Cell & Environment, 29*, 315–330.

Lopes, D. B., & Berger, R. D. (2001). The effects of rust and anthracnose on the photosynthetic competence of diseased bean leaves. *Phytopathology, 91*, 212–220.

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

Macedo, T. B., Peterson, R. K. D., & Weaver, D. K. (2006). Photosynthetic responses of wheat, *Tritcum aestivum* L., plants to simulated insect defoliation during vegetative growth and at grain fill. *Environmental Entomology, 35*, 1702–1709.

Martin, P. J. (1986). Gaseous exchange studies of barley leaves infected with *Rhynchosporium secalis* (Oudem) J.J. Davis. *Physiological and Molecular Plant Pathology, 28*, 3–14.

Matyssek, R., Agerer, R., Ernst, D., Munch, J.-C., Oßwald, W., Pretzsch, H., et al. (2005). The plant's capacity in regulating resource demand. *Plant Biology, 7*, 560–580.

Mauricio, R., Rauscher, M. D., & Burdick, D. S. (1997). Variation in the defense strategies of plants: Are resistance and tolerance mutually exclusive? *Ecology, 78*, 1301–1311.

Meyer, S., Saccardt, A. K., Rizza, F., & Gentry, B. (2001). Inhibition of photosynthesis by *Colletotrichum lindemuthianum* in bean determined by chlorophyll fluorescence imaging. *Plant, Cell & Environment, 24*, 947–955.

Minchin, P. E. H., & Lacointe, A. (2005). New understanding on phloem physiology and possible consequences for modelling long-distance carbon transport. *New Phytologist, 116*, 771–779.

Munier-Jolain, N. G., & Ney, B. (1998). Analysis of seed growth rate for different species of grain legumes. II - Cotyledon cell number and seed growth rate in *Pea (Pisum sativum* L.). *Journal of Experimental Botany, 49*, 1971–1976.

Murray, D., & Walters, D. R. (1992). Increased photosynthesis and resistance to rust infection in upper, uninfected leaves of rusted broad bean (*Vicia faba* L.). *New Phytologist, 120*, 235–242.

Mussell, H. W., & Malone, M. J. (1979). Disease tolerance: reducing the impact of disease-induced stress on crop yields. In H. Mussel, R. C. Staples, & B. Thompson (Eds.), *Stress physiology in crop plants* (pp. 15–23). New York: John Wiley & Sons.

Nabity, P. D., Zavala, J. A., & DeLucia, E. H. (2009). Indirect suppression of photosynthesis on individual leaves by arthropod herbivory. *Annals of Botany, 103*, 655–663.

Neth, J. (1992). Evaluation of breeding strategies for resistance and tolerance to late blight in potato by means of simulation. *Plant Pathology, 98*, 3–11.

Newingham, B. A., Callaway, R. M., & BassiriRad, H. (2007). Allocating nitrogen away from a herbivore: a novel compensatory response to root herbivory. *Oecologia, 153*, 913–920.

Oliver, P. R., & Ipcho, S. V. S. (2004). Arabidopsis pathology breathes new life into the necrotrophs-vs.-biotrophs classification of fungal pathogens. *Molecular Plant Pathology, 5*, 347–352.

Oliver, T. H., Leather, S. R., & Cook, J. M. (2009). Tolerance traits and the stability of mutualism. *Oikos, 118*, 346–352.

Parker, S. R., Welham, S., Paveley, N. D., Foulkes, J., & Scott, R. K. (2004). Tolerance of septoria leaf blotch in winter wheat. *Plant Pathology, 53*, 1–10.

Parry, M., Reynolds, M., Salvucci, M., Raines, C. A., Andralojc, P. J., Zhu, X. G., et al. (2011). Raising yield potential of wheat. II. Increasing photosynthetic capacity and efficiency. *Journal of Experimental Botany, 62*, 453–467.

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

Paveley, N. D., Smith, J. S., & Foulkes, J. (2008). *Traits for reduced fungicide dependence. In Cereal Pathosystems* (pp. 35–47). London: BSPP.

Pinet, A. (2010). Analyse de la plasticité du puits reproducteur du colza suite à une suppression de boutons floraux : conséquences sur le rapport source: puits et le rendement. PhD dissertation. University of Paris. AgroParisTech. 141p.

Reynolds, M. P., Pellegrineschi, A., & Skovmand, B. (2005). Sink-limitation to yield and biomass: a summary of some investigations in spring wheat. *Annals of Applied Biology, 146*, 39–49.

Reynolds, M. P., Calderini, D., Ciondon, A., & Vargas, M. (2007). Evaluating potential genetic gains in wheat associated with stress-adaptive trait expression in elite genetic resources under drought and heat stress. *Crop Science, 47*, 172–178.

Reynolds, M., Manes, Y., Izanloo, A., & Langridge, P. (2009). Phenotyping approaches for physiological breeding and gene discovery in wheat. *Annals of Applied Biology, 155*, 309–320.

Robb, J. (2007). Verticillium tolerance: resistance, susceptibility or mutualism? *Canadian Journal of Botany, 85*, 903–910.

Robert, C., Bancal, M.-O., Nicolas, P., Lannou, C., & Ney, B. (2004). Analysis and modelling of effects of leaf rust and *Septoria tritici* blotch on wheat growth. *Journal of Experimental Botany, 55*, 1079–1094.

Robert, C., Bancal, M.-O., Ney, B., & Lannou, C. (2005). Wheat leaf photosynthesis loss due to leaf rust, with respect to lesion development and leaf nitrogen status. *New Phytologist, 165*, 227–241.

Robert, C., Bancal, M.-O., Lannou, C., & Ney, B. (2006). Quantification of the effects of *Septoria tritici* blotch on wheat leaf gas exchange with respect to lesion age, leaf number, and leaf nitrogen status. *Journal of Experimental Botany, 57*, 225–234.

Robert, C., Fournier, C., Dornbusch, T., Andrieu, B. & Gouache, D. (2010). Using a functional-structural plant model to design wheat ideotypes and assess their performance regarding potentials of biomass production, weed suppression and disease escape. 6th International workshop on Functional - Structural Plant Model. Davis, USA.

Roberts, A. M., & Walters, D. R. (1986). Stimulation of photosynthesis in uninfected leaves of rust-infected leeks. *Annals of Botany, 56*, 893–896.

Rolfe, S. A., & Scholes, J. D. (2010). Chlorophyll fluorescence imaging of plant-pathogen interactions. *Protoplasma, 247*, 163–175.

Rooney, J. M., & Hoad, G. V. (1989). Compensation in growth and photosynthesis of wheat (*Tritcum aestivum* L.) following early inoculations with *Septoria nodurum* (Berk.). *New Phytologist, 113*, 513–521.

Rosenthal, J. P., & Welter, S. C. (1995). Tolerance to herbivory by a stemboring caterpillar in architecturally distinct maizes and wild relatives. *Oecologia, 102*, 146–155.

Rossing, W. A. H., van Oijen, M., van der Werf, W., Bastiaans, L., & Rabbinge, R. (1992). Modelling the effects of foliar pests and pathogens on light interception, photosynthesis, growth rate and yield of field crops. In P. G. Ayres (Ed.), *Pests and Pathogens. Plant responses to foliar attack* (pp. 161–180). Oxford: Bios Scientific Publishers.

Roux, F., Gao, L., & Bergelson, J. (2010). Impact of initial pathogen density on resistance and tolerance in a polymorphic disease resistance gene system in *Arabidopsis thaliana*. *Genetics, 185*, 283–291.

Roy, B. A., & Kirchner, J. W. (2000). Evolutionary dynamics of pathogen resistance and tolerance. *Evolution, 54*, 51–63.

Sadras, V. O. (2000). Variation in apical dominance and its implications for herbivory resistance, competitive ability, and biomasspartitioning. In M. E. Otegui & G. A. Slafer (Eds.), *Physiology Bases for Maize Improvement* (p. 217). Chesterfield: Food Products Press.

Savary, S., Teng, P. S., Willocquet, L., & Nutter, F. W. J. (2006). Quantification and modeling of crop losses: a review of purposes. *Annual Review of Phytopathology, 44*, 89–112.

Schafer, J. F. (1971). Tolerance to plant disease. *Annual Review of Phytopathology, 9*, 235–252.

Schoeny, A., Jeuffroy, M.-H., & Lucas, P. (2003). Influence of take-all epidemics on winter wheat yield formation and yield loss. *Phytopathology, 91*, 694–701.

Scholes, J. D., & Rolfe, S. A. (1996). Photosynthesis in localised regions of oat leaves infected with crown rust (*Puccinia coronata*): quantitative imaging of chlorophyll fluorescence. *Planta, 199*, 573–582.

Scholes, J. D., & Rolfe, S. A. (2009). Chlorophyll fluorescence imaging as a tool for understanding the impact of fungal; diseases on plant performance: a phenomics perspective. *Functional Plant Biology, 36*, 880–892.

Schwachtje, J., & Baldwin, I. T. (2008). Why does herbivore attack reconure primary metabolism? *Plant Physiology, 146*, 845–851.

Schwachtje, J., Minchin, P. E. H., Jahnke, S., van Dongen, J. T., Schittko, U., & Baldwin, I. T. (2006). SNF1-related kinases allow plants to tolerate herbivory by allocating carbon to roots. *Proceedings of the National Academy of Sciences of the United States of America, 103*, 12935–12940.

Serrago, R., Carretero, R., Bancal, M.-O., & Miralles, D. J. (2011). Grain weight response to foliar diseases control in wheat (*Triticum aestivum* L.). *Field Crops Research, 120*, 352–359.

Shaw, M. W., & Osborne, T. M. (2011). Geographic distribution of plant pathogens in response to climate change. *Plant Pathology, 60*, 31–43.

Shtienberg, D. (1992). Effects of foliar diseases on gas exchange processes: a comparative study. *Phytopathology, 82*, 760–765.

Smedegaard-Petersen, V., & Tolstrup, K. (1985). The limiting effect of disease resistance on yield. *Annual Review of Phytopathology, 23*, 475–490.

Spink, J. H., Semere, T., Sparkes, D. L., Whaley, J. M., Foulkes, M. J., Clare, R. W., et al. (2000). Effect of sowing date on the optimum plant density of winter wheat. *Annals of Applied Biology, 137*, 179–188.

Spitters, C. J., van Roermund, H. J. W., van Nassau, H. G. M. G., Scheper, J., & Mesdag, J. (1990). Genetic variation in partial resistance to leaf rust in winter wheat: disease progress, foliage senescence and yield reduction. *Netherlands Journal of Plant Pathology, 96*, 3–15.

Strauss, S., & Aggrawal, A. (1999). The ecology and evolution of plant tolerance to herbivory. *Trends in Ecology & Evolution, 14*, 179–185.

Swarbrick, P. J., Schulze-Lefert, P., & Scholes, J. D. (2006). Metabolic consequences of susceptibility and resistance (race-specific and broad-spectrum) in barley leaves challenged with powdery mildew. *Plant, Cell & Environment, 29*, 1061–1076.

Sylvester-Bradley, R., & Wiseman, J. (2005). *Yields of Farmed Species. In Proceedings of the 61st Easter School in Agricultural Sciences*. UK: Nottingham University Press.

Sylvester-Bradley, R., Riffkin, P., & O'Leary, G. (2012). Designing resource-efficient ideotypes for new cropping conditions: Wheat (*Triticum aestivum* L.) in the high rainfall zone of South Australia. *Field Crops Research, 125*, 69–82.

Tang, J., Zielinski, R., Aldea, M., & DeLucia, E. (2009). Spatial association of photosynthesis and chemical defense in *Arabidopsis thaliana* following herbivory by *Trichoplusia ni*. *Physiologia Plantarum, 1347*, 115–124.

Thomson, V. P., Cunningham, S. A., Ball, M. C., & Nicotra, A. B. (2003). Compensation for herbivory by *Cucumis sativus* through increased photosynthetic capacity and efficiency. *Oecologia, 134*, 167–175.

Tiffin, P. (2000). Mechanisms of tolerance to herbivore damage: what do we know? *Evolutionary Ecology, 14*, 523–536.

Tiffin, P. (2002). Competition and time of damage affect the pattern of selection acting on plant defense against herbivores. *Ecology, 83*, 1981–1990.

Tiffin, P., & Rausher, M. D. (1999). Genetic constraints and selection acting on tolerance to herbivory in the common morning glory Ipomoea purpurea. *American Naturalist, 154*, 700–716.

Vannette, R. L., & Hunter, M. D. (2011). Plant defense theory re-examined: nonlinear expectations based on the costs and benefits of resource mutualisms. *Journal of Ecology, 99*, 66–76.

Waggoner, P. E., & Berger, R. D. (1987). Defoliation, disease and growth. *Phytopathology, 77*, 393–398.

Walters, D. R., & McRoberts, N. (2006). Plants and biotrophs: a pivotal role for cytokinins? *Trends in Plant Science, 11*, 581–586.

West, S. J., Townsend, J. A., Stevens, M., & Fitt, B. D. L. (2012). Comparative biology of different plant pathogens to estimate effects of climate change on crop diseases in Europe. *European Journal of Plant Pathology, 133*, 315–331.

Williams, G. M., & Ayres, P. G. (1981). Effects of powdery mildew and water stress on CO_2 exchanges in uninfected leaves of barley plants. *Plant Physiology, 68*, 527–530.

Wise, M. J., & Abrahamson, W. G. (2005). Beyond compensatory continuum: environmental and plant tolerance herbivory. *Oikos, 109*, 417–428.

Zangerl, A. R., Hamilton, J. G., Miller, T. J., Crofts, A. R., Oxborough, K., Berenbaum, M. R., et al. (2002). Impact of folivory on photosynthesis is greater than the sum of its holes. *Proceedings of the National Academy of Sciences of the United States of America, 99*, 1088–1091.

Zuckerman, E., Eshal, A., & Eyal, Z. (1997). Physiological aspects related to tolerance of spring wheat cultivars to *Septoria tritici* blotch. *Phytopathology, 87*, 60–65.