

Seduced by the dark side: integrating molecular and ecological perspectives on the influence of light on plant defence against pests and pathogens.

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Summary

Plants frequently suffer attack from herbivores and microbial pathogens, and have evolved a complex array of defence mechanisms to resist defoliation and disease. These include both preformed defences, ranging from structural features to stores of toxic secondary metabolites, and inducible defences, which are activated only after an attack is detected. It is well known that plant defences against pests and pathogens are commonly affected by environmental conditions, but the mechanisms by which responses to the biotic and abiotic environments interact are only poorly understood. In this review, we consider the impact of light on plant defence, both in terms of plant life histories and rapid scale molecular responses to biotic attack. We bring together evidence that illustrates that light not only modulates defence responses via its influence on biochemistry and plant development, but in some cases, is essential for the development of resistance. We suggest that the interaction between the light environment and plant defence is multifaceted, and extends across different temporal and biological scales.

Key words:

Light, defence, resistance, tolerance, pathogen, herbivore.

Abbreviations:

CBDC – carbon-based defensive chemicals; CHS – chalcone synthase; CNB – carbon nutrient balance; GDB – growth differentiation balance; HR – hypersensitive response; PAL – phenylalanine ammonia lyase; PAR – photosynthetically active radiation; PET – photosynthetic electron transport; PR – pathogenesis-related; ROS – reactive oxygen species.

I. Introduction

Light is fundamental to the existence of plants. It affects all aspects of growth and development, since a primary requirement for plant fitness is to optimise light harvesting for photoautotrophic growth. Hence, photoreceptors such as phytochromes and cryptochromes sense quantitative and qualitative features of the light environment and, via associated signal transduction pathways, regulate plant physiology and development. Light, through photosynthesis, also controls much of the biochemical activity within plant tissues, something that is reflected by the fact that a wide array of genes are transcriptionally regulated by the circadian clock in *Arabidopsis thaliana* (Harmer *et al.*, 2000). By sensing day length, plants also use light as a seasonal indicator which controls the transition to reproductive growth in many plant species. Although essential, light can also pose problems for plants. Increased doses of UV light can cause damage at the molecular level, and even simple changes in ambient sunlight can over-load photosynthetic electron transport (PET), causing damaging reactive oxygen species (ROS) to accumulate. Plants have evolved many systems to minimise the impacts of such deleterious effects of light, including the production of photoprotective pigments, biochemical systems to rapidly modulate chloroplast electron transport, physiological responses such as the ability to re-orient chloroplasts, and photomorphogenic responses that optimise the interaction of the leaves with light over longer time scales.

As well as direct effects on plant metabolism, growth and development, light inevitably influences many other plant responses to the environment. These include defences against pests and pathogens. There is a wide range of information in the scientific literature on

the effects of light on defence responses, ranging from ecological to molecular scale investigations of both short and long term responses. Our aim here is to draw some general conclusions on the impact of light on plant defence, and to attempt to suggest conceptual models that explain the observations in terms of both the molecular and ecophysiological responses to light and biotic attack.

II. Light as an environmental variable

Light is an extremely dynamic component of the terrestrial environment. Changes are both quantitative (including variation in instantaneous irradiance, dose accumulated over time, and day length) and qualitative (in terms of light spectral balance). Plants and their associated herbivores and pathogens may respond to each of these different components of variation.

1. Variation in the quantity of light.

The quantity of light falling on a surface at a given moment, usually referred to as “light intensity,” is formally defined in terms of either energy per unit area (= irradiance) or quanta per unit area (= photon flux: see Bjorn, 2002). Some elements of the variation in irradiance are predictable, for example variation with time of day, season and latitude are all functions of the elevation of the sun in the sky (the higher the solar elevation, the higher the irradiance). As a result, irradiance reaches a maximum near the equator, at mid-day, and, at mid-high latitudes, in mid-summer. Superimposed on these systematic geographical and seasonal variations in irradiance are variations due to factors like cloud cover, aspect on a sloping site, or shade from nearby structures or plant canopies (Fitter &

Hay, 2002). Some of these factors affect all wavelengths of light more or less equally, others are much more wavelength specific (see below).

Many biological responses to light can be described as simple functions of irradiance. The rate of photosynthesis in plants is a typical example. Although photosynthesis is a function of irradiance, growth is determined by the sum of photosynthetic carbon fixation over time which is, in turn, a function of the amount of light received by the plant over that period. Thus, growth and yield, and many other long-term effects of light, are best described by the accumulated dose of photosynthetic radiation, for example by daily light integral (Kitaya *et al.*, 1998; Korczynski *et al.*, 2002; Dielen *et al.*, 2004). Light damage is also often a function of accumulated dose, as with many whole-plant responses to UV radiation (Gonzalez *et al.*, 1998; de la Rosa *et al.*, 2001).

2. Variation in the quality of light

Light quality is the balance between different wavelengths. Different organisms perceive different wavelengths in different ways. The three primary colours of human vision define “visible” light (approx. 400-700 nm), but other animals, including many invertebrate and some vertebrate herbivores, may perceive different wavebands, notably in the ultraviolet region (primarily UV-A: 315-400 nm). Thus, what is actually perceived as “visible light” varies substantially between species. Photosynthetically active radiation (PAR) is usually defined as 400-700 nm, but plants also detect and utilise different wavelengths as environmental cues. Responses to red and far red (detected by phytochromes), blue and UV-A (detected by cryptochromes, phototropins and related

photoreceptors) are well defined (Gyula *et al.*, 2003; Spalding & Folta, 2005). The mechanistic basis for responses to UV-B (290-315nm) remains poorly defined: some may be a function of damage to DNA and other biological molecules, but there is also evidence for a specific UV-B photoreceptor (Jenkins *et al.*, 2001).

The spectral balance of sunlight in the field is influenced by a range of factors. Temporal changes in the ratio of UV to longer wavelengths are largely driven by the increase in the ratio at high solar elevations. At temperate and high latitudes sunlight is relatively enriched in UV, especially UV-B, in summer compared with winter. Similarly, the ratio of UV to PAR is highest near noon. There is some enrichment of far-red relative to red at twilight (Salisbury, 1981). Spatially, UV:PAR ratios are typically higher at low latitudes. Cloud typically reduces all wavelengths of sunlight but shorter wavelength UV less than PAR, resulting in some increase in UV:PAR ratio under cloud conditions (Calbo *et al.*, 2005). Shade from plant canopies has major effects on spectral balance, notably in terms of R:FR (Ballare, 1999; Gyula *et al.*, 2003; Vandenbussche *et al.*, 2005), but also the ratio of UV:PAR (Grant & Heisler, 2001; Heisler *et al.*, 2003; Grant *et al.*, 2005).

III. Long term effects of light on plant-herbivore or plant-pathogen interactions

Studies of the effects of both shade and diurnal variation in light on plant interactions with their natural enemies deal mostly with herbivores; effects on disease remain relatively poorly known. Studies of herbivory (Table 1) have mostly been in the context of variation in the light environment due to plant canopies, such as the effect of position relative to neighbours, including gaps in woodland canopies, or woodland edge

(individuals within gaps or at the forest edge receiving more sunlight than those within). Studies of woody plants have also considered the influence of vertical position in the canopy (foliage at or near the top of the canopy receiving greater insolation than that low down in the canopy), and the direction in which foliage faces (in the northern hemisphere, south facing foliage receives higher irradiances than north-facing). Experiments have either used these natural variations in light environment (for example taking foliage from, or placing plants in, different locations) or artificially manipulated light using shade cloth *etc.* (Table 1). In some cases, the latter experiments have related to the use of shading as a tool in crop production. Of course, shading results in complex changes in the light environment, both quantitative and qualitative, which can differ depending on the source of shade. Thus, although some studies implicitly assume that shade influences plant-herbivore interactions through changes in photosynthesis driven simply by the reduction in PAR, there may be independent effects of altered spectral balance in shade (R:FR or UV:PAR see above). Artificial shade treatments do not necessarily reproduce these spectral changes.

In the field, shade will also influence overall radiation balance with possible consequences for the abiotic environment of the host, the herbivore and potentially other organisms such as parasitoids or predators of the herbivore. Temperatures of the air and of organisms are typically lower in the shade, with direct effects on a wide range of processes, and indirect effects such as altered water balance, which may result in reduced plant water deficits compared with full sunlight. Equally, “shade” in the field, may alter the biotic environment through mechanisms unrelated to any effect on solar radiation. For

example, there is a substantial literature on the role of tree canopies in determining the number and species richness of the community of insectivorous birds that can have a major influence on herbivory (Marquis & Whelan, 1994; Strong *et al.*, 2000; Van Bael & Brawn, 2005). Certainly the effects of tree canopies on herbivory in crops such as coffee can be interpreted in relation to the greater predation by birds, not changes in the light environment (Perfecto *et al.*, 2004). Such effects highlight the complexity of shade as an environmental variable. Clearly, many of the same arguments can be made in relation to comparisons between day and night, which differ in far more than simply the light environment. While these broader mechanisms are largely beyond the scope of this review, they provide an important context for any assessment of light-mediated changes.

1. Light and herbivory

Day / night

Diurnal variation in herbivory has been viewed primarily as a function of the biology of the herbivore rather than the host. The general expectation is that most invertebrate herbivores are less active during the day than at night, at least partly because the risk of predation or parasitism is greater during the day (Hassell & Southwood, 1978). However, there are many exceptions to this pattern (Springate & Basset, 1996; VanLaerhoven *et al.*, 2003). For example, Novotny *et al.*, 1999) reported a three times greater risk of predation during day compared with night, yet herbivores were more abundant during daylight. Some insect herbivores feed almost exclusively during the day (Kreuger & Potter, 2001), with the temperature dependence of behaviour perhaps being a key driver. One host characteristic that shows diurnal variation and which might influence both herbivores and

higher trophic levels is the emission of volatiles. There are quantitative and qualitative differences in wound-induced volatiles between day and night (De Moraes *et al.*, 2001; Gouinguene & Turlings, 2002; Martin *et al.*, 2003, and see Section V). Herbivores, and their parasites and predators, are able to detect and respond to such changes, and diurnal variation in the volatile signal may result in differential effects on different herbivores (De Moraes *et al.*, 2001) as well as on higher trophic levels (Maeda *et al.*, 2000). On the other hand, some predators appear capable of isolating key information against this highly variable volatile signal (Meiners *et al.*, 2003).

Shade

The general hypothesis that herbivory would be suppressed in plants grown in full sun compared with those in shade has been shown to be correct in many systems, especially, but not exclusively, with leaf chewing insects (Table 1a). This is true at least in the sense that when consumed, leaf tissue from plants grown in shade is more favourable to herbivore growth and/or development. However, plants grown in full sunlight may suffer an increase in the leaf area consumed compared with shade-grown plants (Table 1a). This increased consumption may be a function of reduced food quality in full light, since insects often compensate for low food quality by increasing intake (Slansky & Wheeler, 1992). However, this mechanism may not fully explain increased consumption of high-light tissue, since preferences can persist even when extracts of sun or shade-grown leaves are incorporated into artificial diets (Panzuto *et al.*, 2001). These plant-mediated changes interact with herbivore responses. For example, adults of some insect herbivores may prefer high-light locations, including, for example, for egg laying (Alonso, 1997).

There are clear examples where such direct herbivore responses outweigh greater host quality of shade-grown plants (Sipura & Tahvanainen, 2000). Interestingly, an example where herbivore damage is more severe in plants grown at higher light is one of the few examples where light-dependent variation in herbivory has been proven to have significant effects on host population dynamics (Louda & Rodman, 1996). In that study, the native crucifer *Cardamine cordifolia* suffered significantly greater herbivory when natural shade was removed. Some components of host resistance were reduced in full sun, but many were increased, and some of these changes appeared to be related to the mild water deficits that occurred in plants growing in full sun. Insects were also more abundant in the sun. Overall, changes in herbivory were attributed to the combined effects of changes in host defence (with responses perhaps being partly to light and partly to water deficits) and herbivore abundance (Louda & Rodman, 1996).

Although canopy shade may have slightly different effects on PAR and UV wavelengths (Grant & Heisler, 2001; Heisler *et al.*, 2003; Grant *et al.*, 2005), in broad terms the two are highly correlated across natural gradients between sun and shade. Thus, the great majority of research into the effects of shade on herbivory will have manipulated both PAR and UV, even though the possible role of the UV-component of sunlight is generally ignored in interpreting results. The growing literature on the specific effects of UV wavelengths on plant-herbivore interactions demonstrates that variation in UV, or at least UV-B, can be significant in many systems (Table 2). Indeed, it has been suggested that cyclical variation in the population of both vertebrate and invertebrate herbivores may be driven by the effects of natural variation in solar UV-B on host defensive chemistry

(Selas *et al.*, 2004). The experimental manipulation of UV-B alone results in changes in plant-herbivore interactions that show many parallels with those seen with broad-spectrum shading. In most studies, foliage from reduced UV-B environments is generally found to be a higher quality resource for herbivores than foliage from unfiltered sunlight in terms of herbivore mortality, growth rates or the efficiency of food utilisation (Table 2). In the field, defoliation due to herbivory is often increased when ambient solar UV-B is reduced using wavelength-specific filters (Table 2). However, as with “total shade” treatments, both laboratory and field studies show that these UV effects vary between host species, and perhaps genotype, and also between herbivores (Table 2). The mechanisms by which exposure to UV could directly affect insect herbivores remain rather unclear, although the visual systems of many insects perceive longer wavelength UV. The consequent disruption of foraging and dispersal in UV-deficient conditions can be significant in both experimental studies (Mazza *et al.*, 1999) and in the use of UV-opaque plastics for the control of horticultural pests such as thrips and whiteflies (reviewed by Raviv & Antignus, 2004). In the field, UV might also influence herbivore populations through the suppression of entomopathogens, whether nematodes (Fujie & Yokoyama, 1998), fungi (Braga *et al.*, 2001; Braga *et al.*, 2002), bacteria (Myasnik *et al.*, 2001) or viruses (Shapiro & Domek, 2002).

The extent to which reductions in solar UV contribute to the overall effects of shade on plant-herbivore interactions remains unclear. So far as we are aware, the only study to explicitly consider the effects of both UV and shade is that of (Rousseaux *et al.*, 2004) who studied herbivory of *Nothofagus antarctica*. Both the number of sites attacked and

the area of leaf removed by insect herbivores were reduced on the sun-exposed side of the canopy. This response occurred even when UV-opaque filters removed the UV-B component of sunlight. However, removing UV-B significantly reduced leaf area removed on both sun-exposed and shaded sites. This data suggests that the effects of UV-B and those of other components of natural shade can act independently, a contention that is supported by chemical changes induced (see below).

2 Light and disease

Day / night

Whilst defoliation by many herbivores is sufficiently rapid to differentiate damage occurring during day from that at night, disease is a longer term process. Thus, it is not surprising that, so far as we are aware, investigations of diurnal changes in plant-pathogen interactions have dealt with specific aspects, such as sporulation, spore dispersal or infection. The concentration of air-borne spores in and around plant canopies is far higher at night than during the day in a wide range of fungi (Schmale & Bergstrom, 2004; Gilbert, 2005; Zhang *et al.*, 2005). However, in other fungal pathogens spore concentrations peak during the day (Gadoury *et al.*, 1998; Su *et al.*, 2000) or show more complex diurnal patterns (Hock *et al.*, 1995). These processes in plant-pathogen interactions may be influenced by the lower temperature, higher humidity or the presence of leaf surface water from dew occurring at night and, as with herbivory, it is not always clear what role is played by direct effects of light. However, there is clear evidence that spore release is initiated by light in some systems (Gadoury *et al.*, 1998; Su *et al.*, 2000). Light also directly inhibits spore germination and or germ tube growth in many plant

pathogenic fungi (Elison *et al.*, 1992; Joseph & Hering, 1997; Tapsoba & Wilson, 1997; Mueller & Buck, 2003; Beyer *et al.*, 2004), and this is certainly the case for UV (Paul, 2000). Overall, it is probably the case that plants are subject to greater challenge by many pathogens at night than during the day, but this is certainly not the case for all pathogens.

Shade

The influence of shade on plant-pathogen interactions has been much less studied than comparable effects on plant-herbivore interactions. However, a number of studies of non-crop systems have shown that shade increases infection by a range of pathogens (Table 1b). As with herbivory, there are exceptions to the usual expectation that disease is more severe in the shade, as seen with coffee rust (*Hemileia vastatrix*) (Soto-Pinto *et al.*, 2002), anthracnose (*Colletotrichum gloeosporioides*) of *Euonymus fortunei* (Ningen *et al.*, 2005) and powdery mildew (*Microsphaera alphitoides*) on oak (*Quercus petraea*: Kelly, 2002). For the most part, the mechanisms by which shade influences plant-pathogen interactions remain poorly understood, although plant pathologists have often attributed the effects of shade to factors such as humidity or leaf surface wetness, which are clearly central to the biology of many plant pathogens (Jarosz & Levy, 1988; Meijer & Leuchtman, 2000; Koh *et al.*, 2003). However, a number of studies have shown that infection by a range of pathogens can be affected by the light environment of the host prior to inoculation. While wheat seedlings exposed to low light intensity were more susceptible to subsequent inoculation by *Puccinia striiformis* than dark-grown seedlings (de Vallavieille-Pope *et al.*, 2002), in other cases infection is inversely proportional to pre-inoculation irradiances (Zhang *et al.*, 1995; Shafia *et al.*, 2001). This indicates direct effects of light on host

resistance. Furthermore, Pennypacker, 2000), showed that reduced light led to increased infection by *Sclerotinia sclerotiorum* in soya bean, and *Verticillium albo-atrum* but not *Fusarium oxysporum* in alfalfa. This was linked to host resistance mechanisms since the effects of shade in both crops only occurred in resistant genotypes where resistance was quantitative (requiring a large investment of resources) rather than qualitative (based on the hypersensitive response, requiring a smaller investment of energy (Pennypacker, 2000). These conclusions parallel much thinking concerning herbivore resistance (see below).

Light quality as well as light quantity can affect disease. Red light suppressed powdery mildew of cucumber, and the effect appeared to be reversed by far-red (Schuerger & Brown, 1997). There are also indications that host resistance may be induced by pre-inoculation exposure to red light (Islam *et al.*, 1998; Rahman *et al.*, 2002; Khanam *et al.*, 2005). Pathogenic fungi may respond directly to spectral balance, and this is exploited by the use of plastic films which modify spectral balance as a component of disease control in horticulture. Films which transmit more blue light than longer or shorter wavelengths can be used to suppress sporulation in downy mildews and *Botrytis cinerea* (Reuveni & Raviv, 1992, , 1997). Similarly, many plant pathogens use UV radiation as a cue to regulate sporulation, and films opaque to UV radiation can be used to reduce a wide range of crop diseases (reviewed by Raviv & Antignus, 2004). However, manipulating UV has complex effects on pathosystems. While UV-A may stimulate sporulation, exposed fungal tissues can be vulnerable to UV-B radiation, and solar UV-B is a major constraint on the spore survival of many pathogens (Paul, 2000). The effects of reduced

UV-B may be sufficient to explain the overall increase in disease in shade (Gunasekera *et al.*, 1997) or variation in cloud cover (Paul, 2000; Wu *et al.*, 2000). Equally, prior exposure to UV can affect various components of host resistance. Exposure of the host before inoculation reduced subsequent infection in a range of pathosystems, but there are exceptions (reviewed by Gunasekera *et al.*, 1997; Paul *et al.*, 2000). Increases in infection with increased UV-B have been sometimes attributed to host injury providing sites for colonisation by necrotrophic pathogens (Manning & von Tiedemann, 1995), but it is now recognised that this mechanism is probably confined to UV doses well above the ambient range (Paul, 2000). Contrasting responses between pathosystems are certainly not explained simply on the basis of biotrophic and necrotrophic pathogens. Powdery mildews (Erysiphales) are biotrophic pathogens that grow on leaf surfaces exposed to incident radiation. There are several reports that UV-B exposure reduces powdery mildew infections, both in the laboratory (Willoquet *et al.*, 1996; Paul, 1997), and in the field (Keller *et al.*, 2003). However, exposure to increased UV-B led to increased powdery mildew (*Microsphaera alphitoides*) in oak (Newsham *et al.*, 2000), which is consistent with the greater occurrence of this disease in open sites (Kelly, 2002). Overall, the contribution of UV to shade effects on plant-pathogen interactions is likely to be a function of interactions between the relative effects of UV-A and UV-B on direct damage and spore induction in the pathogen, and host resistance mechanisms.

IV. Mechanisms of responses to the light environment: the whole plant perspective.

As discussed above, the literature on the whole plant biology or ecology of the influence of light on plant-herbivore or plant-pathogen interactions is diverse. Responses are

attributed to a wide range of possible underlying mechanisms not only in the host plant, but also light effects on the herbivore or pathogen, or higher trophic levels. Responses may also be associated with other environmental factors correlated with the light environment, rather than light *per se*. With this broad view of underlying mechanisms of response, light-mediated changes in the host plant are viewed as just one component of many. Furthermore, agronomists, and especially ecologists, consider a wide range of host characteristics as being significant in determining the overall effects of light on herbivory or disease. Chemical traits influencing herbivory include tissue nitrogen chemistry (*e.g.* total N concentration, C:N ratio, protein or amino acid concentration), carbohydrate composition (total carbohydrates or components such as the soluble fraction), or water content. Aspects of morphology and physical properties such as leaf thickness, toughness and the possession of thorns or spines can also be significant for plant-herbivore interactions. In addition, the increase in specific leaf area with increasing shade that is commonly observed across a range of species (*e.g.* Crotser *et al.*, 2003; Curt *et al.*, 2005; Poorter *et al.*, 2006) not only influences leaf physical properties but may also change how herbivores respond to chemical defence by changing the relationship between chemical contents and leaf area or biomass. Changes in host resistance, whether constitutive or induced by attack, certainly play an important role in coupling herbivory or disease to the light environment, but this is certainly not the only significant mechanism.

1. Host quality as a food resource for herbivores or pathogens

In a meta-analysis of studies of the effects of abiotic factors on leaf chemistry (Koricheva *et al.*, 1998), shade appeared to have little consistent effect on total leaf nitrogen

concentration or free amino acid concentration across a wide range of woody plant systems. That analysis was explicitly limited to experimental manipulations of shading, and subsequent studies of this type have shown that shade increases total nitrogen and/or amino acid concentrations in some systems (Crone & Jones, 1999; Hemming & Lindroth, 1999; Moon *et al.*, 2000; Dormann, 2003; Henriksson *et al.*, 2003; Baraza *et al.*, 2004; Moran & Showler, 2005) although not in all (Louda & Rodman, 1996; Rowe & Potter, 2000). Koricheva *et al.*, 1998) did not consider responses to natural variation in light environment, due to position in canopy for example. Such studies frequently show significant decreases in leaf nitrogen under shade (*e.g.* Fortin & Mauffette, 2001, , 2002; Yamasaki & Kikuzawa, 2003). Research into canopy photosynthesis also shows that the distribution of nitrogen in the canopy is in proportion to the distribution of absorbed light, with the result that leaves in high light have high nitrogen concentration and contribute the bulk of canopy carbon fixation (Leuning *et al.*, 1995; dePury & Farquhar, 1997). Exposure to UV-B often increases foliage nitrogen concentration (Hatcher & Paul, 1994; McCloud & Berenbaum, 1999; Lindroth *et al.*, 2000; Warren *et al.*, 2002; Keller *et al.*, 2003; Milchunas *et al.*, 2004) but has no effect in some systems (Salt *et al.*, 1998; de la Rosa *et al.*, 2001; Zavala *et al.*, 2001; Veteli *et al.*, 2003; Zaller *et al.*, 2003) and in others causes decreased foliar nitrogen (Robson *et al.*, 2003).

Koricheva *et al.*, 1998) showed that shading of woody species had highly significant effects on the foliar concentrations of total carbohydrates, non-structural carbohydrates, starch and, to a lesser extent, sugars. This analysis is corroborated by more recent research (Wainhouse *et al.*, 1998; Hemming & Lindroth, 1999; Rowe & Potter, 2000;

Fortin & Mauffette, 2001; Henriksson *et al.*, 2003) and the same response also occurs in herbaceous species (Moran & Showler, 2005). Shade also increases leaf water content (Louda & Rodman, 1996; Henriksson *et al.*, 2003; Moran & Showler, 2005), which may have a major influence on herbivore performance (Henriksson *et al.*, 2003).

2. Mechanical defence: spines, thorns and leaf toughness.

Leaves grown under high light have greater mechanical toughness in a wide range of species (Sagers, 1992; Dudt & Shure, 1994; Bergvinson *et al.*, 1995; Louda & Rodman, 1996; Rowe & Potter, 1996; Henriksson *et al.*, 2003; Martinez-Garza & Howe, 2005), although this is not always the case (Rowe & Potter, 2000). Leaf trichomes typically decrease with shading (Franca & Tingey, 1994; Liakoura *et al.*, 1997; Bentz, 2003) and in tomato, more mites were trapped in the trichomes of leaves grown under high light conditions (Nihoul, 1993). The effect of shade on spines, thorns and prickles is less clear. Fisher *et al.*, 2002) showed that reductions in the density of thorns in the tropical liana, *Artabotrys hexapetalus* growing in shaded sites was due to reduced irradiance rather than spectral quality. Bazely *et al.*, 1991) also showed reduced physical defence (prickles) in *Rubus fruticosus* in shaded sites, though this could not be attributed to light *per se*. Changes in the overall morphology and habit of woody plants under shade, rather than any specific physical defence, appear to be a key factor influencing some vertebrate herbivores (Iason *et al.*, 1996; Hartley *et al.*, 1997).

3. Defensive chemistry

Many ecological studies of the mechanisms by which light influences herbivory (there is little comparable research on pathogens) have been conducted in the context of alternative theories of plant defence, such as the resource availability hypothesis (Coley *et al.*, 1985), growth differentiation balance hypothesis (GDB: Herms & Mattson, 1992) and carbon nutrient balance hypothesis (CNB: Bryant *et al.*, 1983). These hypotheses share in common the principle that plant allocation to defence is a function of competition between end-points (growth, storage, defence) for limited resources, such as photosynthate. A meta-analysis of almost 150 published experimental tests of CNB in woody species (Koricheva *et al.*, 1998) revealed that the basic prediction of the hypothesis that shading would reduce concentrations of “carbon-based defensive chemicals” (CBDCs) was broadly correct. Indeed, shading appeared to have a far stronger influence on such compounds than nitrogen supply, which CNB predicts will be inversely related to defence (Koricheva *et al.*, 1998). When CBDCs were divided into three subgroups, phenylpropanoids, hydrolysable tannins and terpenoids, all three were reduced by shading, with phenylpropanoids showing the greatest response (Koricheva *et al.*, 1998). More recent research confirms that shading reduces concentrations of CBDCs, in herbaceous as well as woody species (Jansen & Stamp, 1997; Crone & Jones, 1999; Hemming & Lindroth, 1999; Rowe & Potter, 2000; Tattini *et al.*, 2000; Briskin & Gawienowski, 2001; Henriksson *et al.*, 2003). In addition, it is now clear that shading may reduce concentrations of a wide range of secondary metabolites, not only of CBDCs, which have been the primary focus of studies associated with testing the CNB hypothesis. Shade reduced cyanogenic glycosides but not CBDCs in *Eucalyptus cladocalyx* (Burns *et al.*, 2002), while in *Prunus turneriana*, shade resulted in a change in the distribution of

cyanogenic glycosides between older and younger leaves (Miller *et al.*, 2004). However, shading did not affect the concentration of defensive amides in *Piper cenocladum* (Dyer *et al.*, 2004). Exposure to UV-B increased cyanogenic alkaloids in some genotypes of *Trifolium repens* (Lindroth *et al.*, 2000) and the effects of UV-B on plant phenolics are now very well established, and are not related to the ideas of resource limitation inherent in the CNB hypothesis. In general, increased exposure to UV-B results in increased concentrations of total phenolics (Bassman, 2004), although there are exceptions (Rousseaux *et al.*, 1998; Salt *et al.*, 1998; Levizou & Manetas, 2001). Specific phenolic compounds may show contrasting responses to UV-B, with flavonoids showing particularly consistent increases (Lavola *et al.*, 1998; Tegelberg & Julkunen-Tiitto, 2001; Warren *et al.*, 2002; Lavola *et al.*, 2003; Tegelberg *et al.*, 2003; Warren *et al.*, 2003; Rousseaux *et al.*, 2004), with well established dose responses in some cases (de la Rosa *et al.*, 2001).

Of course, it is certainly not the case that low light reduces the concentration of defensive chemicals in all plants (Burns *et al.*, 2002), and a fundamental point is that not all compounds decline in concentration under low light. This specificity in the effect of shading, and its relationship to the responses of herbivores to putative defensive compounds has been the subject of intense discussion in the context of alternative defence theories (Lerdau *et al.*, 1994; Berenbaum, 1995; Hamilton *et al.*, 2001; Close & McArthur, 2002; Koricheva, 2002; Nitao *et al.*, 2002). Specificity is best characterised for phenolic compounds in woody species. For example, in *Populus tremuloides*, low light reduced proanthocyanidins (condensed tannins) but had less effect on phenolic

glycosides, which were the main factor influencing herbivory (Hemming & Lindroth, 1999). In *Betula pubescens*, total phenolics and soluble proanthocyanidins were reduced by shade netting treatments, but gallotannins (hydrolysable tannins), cell-wall-bound proanthocyanidins and flavonoids (including kaempferols and quercetins) were not affected (Henriksson *et al.*, 2003). The phenolic composition of another birch species (*Betula pendula*) is influenced by light spectral quality. Tegelberg *et al.*, 2004) concluded that increasing R:FR shifted the balance of phenolics from chlorogenic acids to flavonoids, and that this effect was distinct from those of increasing UV-B, which increased concentrations of many flavonoids (kaempferols and quercetins) and chlorogenic acids. Spectral modification had no effect on proanthocyanidins in *Betula pendula* (Tegelberg *et al.*, 2004), unlike shading treatment in *Betula pubescens* (Henriksson *et al.*, 2003). Increased R:FR increased total phenolics in seedlings of *Impatiens capensis* (Weinig *et al.*, 2004), although both these authors and Tegelberg *et al.*, 2004) linked changes in phenolics with the reduced growth observed at higher R:FR. In *Nothofagus antarctica*, removal of solar UV-B radiation increased the concentration of hydrolysable tannins (gallic acid and its derivatives) but decreased the concentration of a flavonoid aglycone (Rousseaux *et al.*, 2004). Flavonoid aglycone was also increased on the sun-exposed side of the canopy, as was quercetin-3-arabinopyranoside (Rousseaux *et al.*, 2004).

The responses of herbivore to shade-induced change in host chemistry are less well explained by bulk chemistry (total phenolics for example), than concentrations of specific compounds (Crone & Jones, 1999; Ossipov *et al.*, 2001; Henriksson *et al.*, 2003;

Lahtinen *et al.*, 2004; Rousseaux *et al.*, 2004). Overall, it is increasingly clear from the ecophysiological literature that the responses of defence-related chemicals to shade are far more subtle than can be explained by the bulk diversion of carbon into secondary metabolism that is predicted by the CNB hypothesis. The molecular and cellular literature is now beginning to shed light on some of the underlying mechanisms through which this fine-tuning of plant secondary metabolism is controlled (see Section VII).

V. Short-term responses to the light environment – induced defences

In addition to the constitutive defences produced by plants that can be influenced by light, evidence is accumulating that induced defences may also be affected. Induced defences are those which involve rapid changes in biochemistry and gene expression in response to herbivore attack or pathogen infection. In the case of pathogen infection, such responses usually require molecular recognition events, such as classic gene-for-gene based resistance. Physical damage can also be sufficient to activate some responses, especially in the case of herbivore defence, although several elicitors of specific responses have been isolated from herbivore oral secretions. The term “induced resistance” broadly refers to plant responses such as the hypersensitive response (HR), the biosynthesis of defensive secondary metabolites (*e.g.* phytoalexins), and the up-regulation of expression of defence genes (such as those encoding pathogenesis-related (PR) proteins and protease inhibitors).

1. Pathogens

There is anecdotal evidence that the development of plant resistance to microbial pathogens can often require illumination during the infection process. The scientific literature contains a number of reports confirming this idea. For example, light is necessary for development of resistance responses to *Pseudomonas solanacearum* in tobacco (Lozano & Sequeira, 1970), *Xanthomonas oryzae* in rice (Guo *et al.*, 1993), and *P. syringae* and *Peronospora parasitica* in Arabidopsis (Mateo *et al.*, 2004; Zeier *et al.*, 2004). Furthermore, red light treatments were able to induce resistance to *Botrytis cinerea* and *Alternaria tenuissima* in broad bean (Islam *et al.*, 1998; Rahman *et al.*, 2003). As well as these studies on interactions between plants and pathogens, there are also several examples of plant responses to isolated pathogenic elicitors that are also light-dependent. For example, leaf necrosis in tomato in response to an avirulence elicitor from *Cladosporium fulvum* is substantially reduced in the dark (Peever & Higgins, 1989), and cell death induced by the fungal toxins AAL from *Alternaria alternata* (Moussatos *et al.*, 1993) and fumonisin B1 (Asai *et al.*, 2000; Stone *et al.*, 2000) requires light, as does the fumonisin B1-induced expression of the SAR marker gene, *PR1* (Asai *et al.*, 2000). In addition, necrotic lesion formation activated by over-expression of the tomato *Pto* disease resistance gene also requires light, although the same authors found that HR mediated by the endogenous *Pto* gene in plants inoculated with an incompatible strain of *P. syringae* was light-independent (Tang *et al.*, 1999). This contrasts with the light-dependence of resistance to the same pathogen in Arabidopsis conferred through a different resistance-avirulence gene interaction (Zeier *et al.*, 2004). Interestingly, programmed cell death caused by UV-C treatment also requires illumination with white light following a lethal

UV-C dose in *Arabidopsis* (Danon *et al.*, 2004). It is important to note, however, that in addition to these examples, there are many inducible defence responses that are clearly not light-dependent. Indeed, responses to the same stimuli can involve light-dependent and independent elements. For example, whereas cell death in response to *C. fulvum* elicitor in tomato was light-dependent, lipoxygenase enzyme activation was not (Peever & Higgins, 1989). Finally, it should be noted that these findings tend to be rather *ad hoc* and based on light/dark differences – very few studies have considered the qualitative or quantitative effects of light on resistance.

In green tissues, chloroplasts are an obvious target that can respond to changes in the light environment, although chloroplasts might not be considered an obvious part of a defence response. However, links between chloroplast function and disease resistance have been identified in several systems. For example, silencing of the 33K subunit of the oxygen-evolving complex of photosystem II (Abbink *et al.*, 2002), or over-expression of the DS9 chloroplast metalloprotease (Seo *et al.*, 2000), both increase susceptibility of tobacco plants to TMV infection. White leaves of the variegated *albostrians* barley mutant support increased growth of the fungal pathogen *Bipolaris sorokiniana* (Schäfer *et al.*, 2004) and fail to produce SA in response to powdery mildew infection (Jain *et al.*, 2004). In *Arabidopsis*, the presence of functional chloroplasts is also required for HR in leaves infected with an incompatible strain of *P. syringae* (Genoud *et al.*, 2002). Thus, resistance in a number of different plant-pathogen interactions requires chloroplast function, though this does not necessarily mean that it requires light.

2. Herbivores

In contrast to pathogen defence, there are relatively few specific studies on the influence of light on induced resistance against herbivores or responses to wounding. One exception to this is the class of so-called indirect defences. These involve the generation of complex mixtures of volatile compounds that are used by predators and insect parasitoids, such as parasitic wasps, as cues to locate their prey or hosts respectively (Paré & Tumlinson, 1999). As noted above, many investigations of herbivore-induced volatile production have shown that this response is largely light-dependent (*e.g.* Loughrin *et al.*, 1994; Halitschke *et al.*, 2000; Maeda *et al.*, 2000; Gouinguene and Turlings, 2002). In general, volatile emission induced by herbivore feeding or by application of methyl jasmonate appears to follow a diurnal cycle, with emission being much stronger during the light period than the dark. However, other defence-related volatiles are also produced during the night (*e.g.* De Moraes *et al.*, 2001).

The plant hormone jasmonic acid (JA) plays a central role in controlling responses to wounding and herbivore attack and to infection by some pathogens, especially necrotrophic fungi. The early steps of JA biosynthesis occur in the chloroplasts of wounded leaves (Turner *et al.*, 2002), but JA synthesis is not necessarily light-dependent. Wound-induced JA biosynthesis was observed in soybean hypocotyls in the dark (Creelman *et al.*, 1992) and also occurs in non-photosynthetic tissues such as potato tubers (Koda & Kikuta, 1994). Furthermore, Zeier *et al.* (2004), observed that pathogen-induced JA levels in *Arabidopsis* were higher in the dark than in the light. This suggests that induced responses to wounding might be largely light-independent, though it is

important to note that in the vast majority of studies, no direct comparison has been made between the wound-induced accumulation of JA under different light conditions, nor, importantly, in the responses to wounding or JA. Where such comparisons have been made, there is evidence in some cases that wound and JA-induced responses can in fact be light-dependent. Most notable amongst these are the indirect defences, but direct defence responses can also be light-dependent. For example, in a series of reports on the expression of stress-inducible genes from rice, several were identified which in general, required light for their induction by wounding and by exogenous JA application (Agrawal *et al.*, 2002a,b,c, 2003). In Arabidopsis, the *ASCORBATE PEROXIDASE 2 (APX2)* gene, is also wound-induced, but by a JA-independent pathway. Instead, it appears to be regulated by changes in photosynthetic electron transport (PET) in wounded leaves, which results in increased levels of ROS (Chang *et al.*, 2004). Interestingly, most of the light-dependent wound-induced genes from rice are also responsive to applied H₂O₂ and copper (a ROS generator), even in the dark (Agrawal *et al.*, 2002b,c, 2003). These data suggest that light-driven generation of ROS in chloroplasts around sites of wounding might be responsible for the expression of a sub-set of wound-induced genes.

VI. Mechanisms for light-dependent induced defences.

Whilst there has been a large body of research defining the physiological basis for the light-dependence of constitutive defences, the basis behind the affect of light on induced resistance is less well understood. There are two general mechanisms by which light could regulate defence responses in plants. The first of these is based on the energetic status of light-driven chemical reactions (dependent on the ability of PET to generate

ATP and reducing power), and the second, the direct perception of light and downstream light-responsive signalling pathways.

1. Photosynthesis and ROS

Photosynthesis uses light energy to drive electrons through complex electron transport chains in the thylakoid membranes, which harvest the energy from activated carriers to ultimately generate ATP and reducing power in the form of NADPH. These key metabolites are then used in carbon fixation in the Calvin cycle, as well as in various other metabolic reactions that take place in the chloroplasts, such as fatty acid biosynthesis and assimilation of nitrogen into amino acids. There are two ways in which these light-dependent processes in chloroplasts could impact on short term, induced defence responses. First, major changes in gene expression, protein synthesis and defence metabolism could potentially be affected by the loss in the dark of substrates synthesized in chloroplasts. Interestingly, at least part of the biosynthetic pathways for three major defence-related hormones, JA, SA and ABA are also located in plastids. Second, as indicated above, chloroplasts can be a significant source of ROS during stress conditions. Plant leaves acclimate to average ambient light intensities during their growth, such that the levels of light harvesting complexes and Calvin cycle enzymes are optimised to make most efficient use of the available light. However, when light intensities transiently increase, or when carbon fixation is prevented, PET generates more electrons than can be accepted by the available electron acceptor NADP⁺. In these situations, free electrons from the electron transport chain can be transferred directly to oxygen to form ROS. Secondly, increased excitation energy can be dissipated via photorespiration, which

ultimately results in the generation of H₂O₂ in the peroxisomes. Normally, a range of biochemical and physiological systems to minimise over-reduction of the electron transport chain and to scavenge those ROS that are produced. However, under severe acute stress, ROS can accumulate to levels that exceed the chloroplast's array of antioxidant systems (Apel & Hirt, 2004). Additionally, damage to the chloroplasts or disruption of chlorophyll biosynthesis can result in the accumulation of photosensitive pigments that can directly generate ROS in the light. Since ROS are well known as important regulators of several defence responses (Apel & Hirt, 2004), significant perturbations in redox balance in the chloroplasts may contribute to ROS-regulated defence.

The implications of the requirement for light for chloroplast-derived ROS may extend beyond the direct signalling roles of ROS. For example, one consequence of ROS production under stress conditions is lipid peroxidation. Many of the products of lipid peroxidation reactions that occur following wounding or pathogen attack, are also reactive electrophile species - molecules with reactive (electrophilic) carbonyl groups (Vollenweider *et al.*, 2000). Many of these electrophiles are now known to act as important signalling molecules, eliciting a range of defence responses ranging from cell death to defence gene expression (Vollenweider *et al.*, 2000; Alméras *et al.*, 2003; Thoma *et al.*, 2003; Cacas *et al.*, 2005). Electrophiles produced as a consequence of stress may either be derived from direct attack of ROS on membrane lipids, or from the activity of lipoxygenase enzymes. Light is therefore likely to directly influence the generation of ROS-derived electrophiles (and downstream responses), but not those generated by

lipoxygenase activity. Interestingly, such effects have been noted in several interactions between plants and pathogens or their elicitors. For example, Montillet *et al.*, (2005) found that in response to the elicitor, cryptogein, cell death was mediated by light-dependent ROS in the light, but in the dark, cell death was independent of ROS and correlated with the activity of a specific lipoxygenase activity. Hence, different mechanisms for the production of bioactive electrophiles may be required to operate under different light environments.

2. Photosensitive pigments and ROS

During pathogen resistance responses, the primary source of ROS is not the chloroplast, but an enzyme found in the plasma membrane known as NADPH oxidase, or respiratory burst oxidase (Apel & Hirt, 2004). One might therefore assume that light-dependent, chloroplast-derived ROS are not likely to be important in pathogen defence. However, the situation is not necessarily clear-cut, since the importance of the NADPH oxidase does not preclude an additional role for chloroplast ROS. Many researchers have isolated mutants from various species, collectively termed lesion mimic mutants, that display spontaneous formation of necrotic lesions on their leaves (Lorrain *et al.*, 2003). These lesions are similar to those formed during the hypersensitive response (a key component of disease resistance responses) and are generally accompanied by the increased expression of PR genes and increased resistance to infection. Generally, lesion mimic mutants were isolated and characterised as part of an effort to understand the mechanisms of disease resistance signalling. However, it is likely that in many cases, these mutants in fact highlight a more general link between chloroplast ROS and plant stress responses,

including pathogen resistance. This idea is discussed in detail elsewhere by Mullineaux and colleagues (Karpinski *et al.*, 2003; Bechtold *et al.*, 2005), but is based on two findings. First is the observation that lesion formation in many of these mutants is light-dependent (*e.g.* Johal *et al.*, 1995; Genoud *et al.*, 1998; Brodersen *et al.*, 2002). Second, cloning of several of the genes defined by these mutations has identified a number of genes involved in chlorophyll biosynthesis or degradation (*e.g.* Hu *et al.*, 1998; Ishikawa *et al.*, 2001; Mach *et al.*, 2001; Pružinska *et al.*, 2003). In addition, manipulation of the expression of several other genes involved in chlorophyll biosynthesis also results in light-dependent lesion mimic phenotypes and increased disease resistance (*e.g.* Kruse *et al.*, 1995; Mock & Grimm, 1997; Mock *et al.*, 1999; Molina *et al.*, 1999). The most likely explanation for these observations is that reactive oxygen species are produced by the action of light on chlorophyll intermediates that act as photosensitizers – that is, they absorb light energy which excites electrons that are subsequently transferred to molecular oxygen to form ROS. These ROS then act as signals to initiate plant defence responses, including pathogen resistance.

Clearly then, the light-dependent generation of ROS from free photosensitive pigments or those present in the photosynthetic light harvesting complexes can impact on defence in mutants and transgenic plants with altered chloroplast biology. The question, then, is whether they do so under normal circumstances. At present, it is not possible to answer this question, but it is likely that plants have evolved mechanisms to deal with the problems of light-dependent ROS generation in tissues under attack from pests and pathogens. For example, the *Arabidopsis* *CHLOROPHYLLASE 1* (*AtCHL1*) gene is

involved in chlorophyll degradation, and is required to remove photosensitive porphyrin ring intermediates. *AtCHLI* is induced by wounding and infection with necrotrophic pathogens (Benedetti *et al.*, 1998; Kariola *et al.*, 2005), at which time it functions to prevent accumulation of ROS generated from breakdown products of chlorophyll released from damaged chloroplasts. Plants with reduced *AtCHLI* gene expression show increased resistance to *Erwinia carotovora*, a necrotrophic bacterial pathogen, but increased susceptibility to *Alternaria brassicicola*, a fungal necrotroph (Kariola *et al.*, 2005). Resistance to *E. carotovora* is conferred by an SA-dependent pathway, whilst resistance to *A. brassicicola* is normally regulated via JA-dependent signalling. Since ROS can potentiate SA-dependent defences which in turn can antagonise JA-dependent resistance, it appears that *AtCHLI* might modulate the balance between SA- and JA-dependent resistance pathways by controlling ROS generation from chlorophyll metabolites. Interestingly, over-expression of the *ACD2* red chlorophyll catabolite reductase gene in Arabidopsis, which would be expected to reduce the accumulation of photosensitizers, generated increased tolerance to a virulent strain of *P. syringae* (Mach *et al.*, 2001). In these plants, bacterial growth was not affected, but cell death symptoms were reduced.

Whilst beyond the scope of this review, it is also notable that many plant species synthesize photosensitizers that are thought to act as direct defences. In the presence of UV-B or white light, these so-called phototoxins generate ROS that function to directly inhibit herbivore or pathogen function (Downum, 1992). Conversely, several genera of fungal pathogens also produce photosensitive toxins, such as cercosporin, that result in

plant cell necrosis (Daub & Ehrenshaft, 2000).

3. Light signalling

The second major mechanism suggested above by which light may regulate defence is via direct light-responsive signalling pathways. Evidence for this type of regulation has been recently uncovered in *Arabidopsis*. Genoud *et al.* (1998) identified an *Arabidopsis* light signalling mutant, *psi2*, that in addition to effects on light-dependent expression of photosynthetic genes, displayed light-dependent development of spontaneous necrotic lesions and increased *PR1* gene expression. Further characterisation of these phenotypes showed that light regulated the resistance responses at multiple levels. First, *PSI2* is a regulator of phytochrome-mediated responses, and *PhyA* and *PhyB* are also required for light-dependent HR lesion formation and PR gene expression (Genoud *et al.*, 2002). Consequently, resistance to *P. syringae* is reduced in phytochrome mutants and increased in the *psi2* mutant. This illustrates an example of light acting in a direct signalling role to modulate induced resistance. How and why phytochrome signalling might impact on disease resistance is unclear, though it might represent a sensitive mechanism by which cytosolic and nuclear responses are matched with changes in chloroplast activity caused by variations in light intensity. Perhaps significantly, in these experiments, HR (although not PR gene expression) also required the presence of functional chloroplasts, since cell death was not observed in white sectors of variegated leaves. Hence, both metabolic and signalling roles for light may combine to co-ordinate a full resistance response.

In terms of induced defences, therefore, we can identify a range of different levels of interaction between light and responses to biotic attack. These include a range of effects on ROS generation, as well as direct signalling roles for light via phytochrome signalling, and are summarised in Figure 1.

VII. Interpreting interactions between light and defence responses.

In assessing the range of experimental systems discussed above, a general conclusion is that where light has been found to modulate plant defence against herbivores or disease, then its effect is usually to increase defence. A key question, therefore, is whether we can identify mechanistic explanations for this observation. As is often the case, ecologists and molecular biologists have taken very different approaches to the question of interactions between light and defence. Given that this is a complex interaction with different components, it is not surprising that such different approaches are possible. Clearly, the fundamental importance of light for plant growth and development means that there is no single explanation that can unite observations across widely different scales of organisation. However, one way forward is to place the whole range of evidence, from molecular to ecophysiological, within the framework of optimal defence theory (Hamilton *et al.*, 2001). Is a greater investment in defence in high light consistent with optimal defence theory, and, if so, does the molecular and cellular data provide insights into the mechanisms through which optimal defence is achieved? This relates to a second important point which is the precise terminology used to describe defence. The semantics of defence in plant pathogen or plant herbivore interactions, which has been widely debated by ecologists and ecophysiologicalists (Clarke, 1986; Stowe *et al.*, 2000), but less so

by cell and molecular biologists, forms a pertinent background to these questions. Defence is defined as any mechanism that protects the plant from reductions in fitness in the presence of herbivores or pathogens and has two components. The first component is resistance, which reduces the severity of attack by inhibiting the activity or performance of the herbivore or pathogen. The second component is tolerance, which reduces the negative consequences of attack on host fitness. In our view the clear differentiation between resistance and tolerance is essential to understanding mechanisms of interactions between light and defence.

The first requirement of optimal defence theory, that tissues which have the greatest value to the plant should be most defended, is clearly satisfied. Models of canopy photosynthesis are consistent in showing that leaves exposed to high light contribute most photosynthate (Leuning *et al.*, 1995; dePury & Farquhar, 1997). Secondly, defence should be in proportion to the probability of attack. There are clearly many systems in which herbivores are more abundant and/or more active in high-light environments, for example due to higher temperatures (see section III). Arguably, the higher nitrogen concentration of high light tissues may increase their potential palatability for herbivores, and so increase the risk of attack. There are certainly examples where exposed tissues suffer more herbivory even though they are better defended (*e.g.* Louda & Rodman, 1996; Sipura & Tahvanainen, 2000). These arguments are harder to apply for pathogens, and if anything, it might be expected that the probability of infection might be lower under high light conditions due partly to direct light effects (see Section II) and partly to the correlated lower humidity and leaf surface water. The third requirement of optimal

defence theory is that defence is a function of the balance between its benefits and its costs. The “broad-brush” prediction of the CNB hypothesis, that defence is less costly under high light conditions because substrates are more freely available, fails to explain the specificity in the responses of individual metabolites to the light environment. Nonetheless, there are a number of other mechanisms that could result in altered costs of defence under different light conditions.

One element of changed costs of defence may relate to the induction of shade-avoidance mechanisms under low light conditions. The possible trade-offs between defence and shade avoidance responses at low light as they relate to competitive ability has recently been reviewed by Cipollini, 2004), who argued that shade avoidance responses could constrain defence via a number of mechanisms. Firstly, the shift in allocation to extension growth under shade might directly compete with allocation to defence, although not necessarily by competition for resources. There may be direct interference between the signalling mechanisms controlling acclimation to the light environment and those regulating defence. Increased stem elongation in the shade response is under the control of auxins and gibberellins (Vandenbussche & Van Der Straeten, 2004). Auxin may interact with defence via cross-talk between IAA and defence signalling, such that IAA reduces JA-induced production of defence compounds (Kernan & Thornburg, 1989; Baldwin *et al.*, 1997). Conversely, the levels of active auxins and the expression of auxin response genes are reduced by wounding (Thornburg & Li, 1991; Cheong *et al.*, 2002; Schmelz *et al.*, 2003) and herbivory (Schmelz *et al.*, 2003). Cipollini, 2004) also suggested that cell wall stiffening might be a mechanism for antagonism between shade

avoidance and defence, with the gibberellin-mediated cell wall loosening leading to increased cell expansion in the shade being incompatible with the cell-wall stiffening that can be a significant component of defence.

Cipollini (2004) described the interference between the shade-response and defence as an opportunity cost but equally, there may be a range of “opportunity benefits” that reduce the cost of defence in high light, because processes induced for photoprotection also confer protection against biotic attack. High light stress, including UV-B irradiation, activates molecular responses that have much in common with pathogen and herbivore responses (Mackerness *et al.*, 1999; Rossel *et al.*, 2002; Kimura *et al.*, 2003; Izaguirre *et al.*, 2003; Stratmann, 2003). In fact, the increasing documentation of the kinds of responses induced by various biotic and abiotic stresses makes it clear that there are many overlaps in these responses. To try to understand the significance of these overlapping responses, it is useful to consider what the functions of induced responses to these different environmental factors might be. For example, many stress responses include increases in the accumulation of antioxidants and the expression of protective chaperone proteins (such as heat shock proteins and osmoprotective proteins). Many forms of environmental insult will disrupt biochemistry leading to increased ROS generation for example, requiring increased antioxidant production to counteract their cytotoxic effects. While there may be many mechanisms for “opportunity benefits”, in our view, many may be based on the involvement of ROS in responses to light, herbivory and disease. Understanding these potential mechanisms requires careful differentiation between resistance and tolerance.

Light and the resistance components of defence against herbivore or pathogen attack

Light-driven generation of ROS in damaged plants may be central to interactions between light and the resistance components of defence against pathogens or herbivores. Photosensitive chlorophyll degradation intermediates formed as a result of cellular damage caused by herbivores and necrotrophic pathogens can contribute to ROS generation and defence signalling (Kariola *et al.*, 2005), as does excess hydrogen peroxide derived from photorespiration (*e.g.* Champognol *et al.*, 1998; Mateo *et al.*, 2004). Several studies described in Section V also indicate a requirement for functional chloroplasts to activate the HR during pathogen resistance, which might also suggest a functional relationship between light-driven reactive oxygen chemistry and defence. NADPH oxidase is clearly an important source of ROS for defence signalling, but is metabolically costly (in terms of NADPH consumption). It is possible that in some systems, ROS generation is supplemented by the action of light on photosensitive pigments such as chlorophyll. Potentially, ROS provides a basis for a “supply side” hypothesis very different from CNB. Resistance is facilitated in (high) light tissue because ROS for signalling can be supplied at less cost via light-driven reactions than those occurring in the dark. Interestingly, there is evidence that elevated UV-B can enhance wound-induced defensive chemicals (Levizou & Manetas, 2001).

There are also specific examples of proteins involved in both resistance and responses to light that may be directly involved in signalling cross-talk. The zinc finger transcriptional

regulator, LSD1 is an Arabidopsis protein first identified through a genetic mutation which conferred a runaway cell death phenotype (Jabs *et al.*, 1996). The *LSD1* gene has been studied mainly with regard to its role as a negative regulator of pathogen-induced hypersensitive cell death. More recently, however, it has also been shown that *LSD1* is also involved in acclimation to high light stress (Mateo *et al.*, 2004). Interestingly, the same authors showed that the effects of *LSD1* on pathogen-induced cell death are mediated by ROS generated during light-dependent photorespiration. NPR1/NIM1 is another signalling protein identified as a key regulator of multiple pathogen resistance pathways. Over-expression of a rice *NPR1* gene leads not only to elevated disease resistance, but also to hypersensitivity to light (Chern *et al.*, 2005).

Light and the tolerance components of defence against herbivore or pathogen attack

As noted above, both biotic attack and light stress are sources of oxidative stress in plant tissues. Furthermore, light and biotic attack may also act synergistically to increase oxidative stress. Biotic stress can result in uncoupling of the light and dark reactions of photosynthesis, meaning that “normal” ambient light levels cause ROS generation from photosynthesis (Bechtold *et al.*, 2005). One common feature of many stress responses is the down-regulation of genes encoding many components of the photosynthetic machinery (*e.g.* Izaguirre *et al.*, 2003; Kimura *et al.*, 2003). This may serve as a negative feedback loop to reduce ROS generation, but also to shift metabolism into areas that compete with photosynthesis, such as the oxidative pentose phosphate and shikimic acid pathways (Scharte *et al.*, 2005). Plant mechanisms involved in protection against oxidative stress or repairing the damage it causes are known to be activated by both light

and herbivore or pathogen attack (*e.g.* Rossel *et al.*, 2002; Kimura *et al.*, 2003; Apel & Hirt, 2004). A key point is that these are tolerance mechanisms not resistance. Clear differentiation between such mechanisms and resistance (*i.e.* mechanisms that inhibit the herbivore or pathogen) is central to understanding interactions between light and defence, not least the widely discussed role of phenolic compounds in such interactions.

Plant phenolics are a highly diverse group of chemicals that fulfil a range of functions. Some phenolics have demonstrable roles in plant interactions with herbivores or pathogens, either as components of resistance (see above) or as attractants for herbivores (*e.g.* Roininen *et al.*, 1999; Ikonen *et al.*, 2002). Other phenolics function as action as “sunscreens” or antioxidants, and some authors have argued that photoprotection is the primary role of many plant phenolics (Close & McArthur, 2002). In considering interactions between light and defence, key points are (i) that plants in high light conditions are potentially confronted with the risk of increased herbivory (see above) and the concurrent need for photoprotection and (ii) that both light and attack can induce oxidative stress. Under such conditions phenolic compounds might fulfil at least three functions: (a) sun-screens reducing light penetration to vulnerable tissues (not selected for by herbivory or disease), (b) antioxidants involved in reducing the damage caused by ROS (selected for by biotic attack as well as light) and (c) resistance compounds inhibiting the activity of herbivore or pathogen (not selected for by light).

These multiple functions would be expected to result in the compound-specific changes in the concentration of phenolics evident in the recent ecophysiological literature (see Section IV). They would also be expected to lead to different trade-offs in the production

of phenolics. In terms of tolerance, the production of phenolic antioxidants in high light tissue might be seen as an opportunity benefit for defence against biotic attack. Conversely, the synthesis of phenolics conferring resistance (*sensuo stricto*) against herbivory or disease may represent an opportunity cost on the production of phenolics acting as sun-screens, and *vice versa*.

The different trade-offs discussed above might be expected to be reflected in enzyme activity and gene expression. From this perspective the three functions of phenolics noted above, while distinct, might all be expected to be associated with an elevated basal flux through the phenylpropanoid pathway. This may explain some of the parallels in terms of global gene expression between herbivory and light stress (*e.g.* Izaguirre *et al.*, 2003; Gachon *et al.*, 2005). Most commonly, it is the genes encoding the enzymes controlling entry of substrates into the phenylpropanoid pathway, such as phenylalanine ammonia lyase (PAL) and chalcone synthase (CHS) that are noted as responsive to multiple stresses. However, such induction of PAL or CHS is clearly only one element in the regulation of the phenylpropanoid pathways and there are examples of competition between elements of phenylpropanoid metabolism delivering compounds with different functions. In *Sorghum bicolor* there is competition between the accumulation of anthocyanin in response to light and the synthesis of phytoalexins in response to challenge by the fungus *Cochliobolus heterostrophus* (Lo & Nicholson, 1998). This was attributed to the down-regulation of genes specific to anthocyanin biosynthesis and the corresponding up-regulation of genes encoding enzymes involved in phytoalexin synthesis (Lo & Nicholson, 1998). Similarly, in grapes, there appears to be competition

between the production of anthocyanins of photoprotection and phytoalexins (resveratrol) for defence against pathogens (Jeandet *et al.*, 1995).

These results show that the plant is able to “fine-tune” phenolic metabolism as the balance of costs and benefits shift in the face of competing end-points. Recent detailed analyses are revealing the details of the regulation of the phenylpropanoid pathway. In the field, exposure of *Vaccinium myrtillus* to full sunlight up-regulates a whole series of phenylpropanoid pathway enzymes but changes in PAL and CHS are much smaller than changes in “downstream” enzymes involved in the synthesis of specific photoprotective compounds (Jaakola *et al.*, 2004). It is clear that sets of several phenylpropanoid pathway genes, for example those involved in flavanol or monolignol biosynthesis, are co-regulated during both development and stress responses (Gachon *et al.*, 2005). In the case of light-responsive expression of flavanol biosynthesis, one mechanism for this co-regulation was demonstrated to stem from the possession of common transcription factor binding sites in the promoters of co-regulated genes (Hartmann *et al.*, 2005). However, while there is clearly co-regulation of major elements of the phenylpropanoid pathway, not all enzymes are represented in these gene expression clusters (Gachon *et al.*, 2005). Furthermore, many key downstream enzymes exist in different isoforms with different substrates and products, fulfilling different functions (Kumar & Ellis, 2003). Thus, up-regulation of a single enzyme, or even a cluster of co-regulated elements of a pathway under high light or biotic attack may reveal little without understanding the behaviour of those enzymes controlling pathway endpoints.

In our view, there is no single answer to the question of how light alters the cost of defence against herbivory or pathogen attack. However, on the balance of the evidence, it seems likely that costs will often become lower with increasing light. This, taken with the greater value of high light tissues and the greater risk of attack, at least by herbivores, suggests that the greater defence is consistent with the predictions of optimal defence theory. The argument that plants have fine control of defence metabolism, which is a major contrast to “supply-side” theories such as CNB, is well-established (*e.g.* Berenbaum, 1995), and molecular studies are increasingly revealing the nature of such fine control. Research at the scale of the transcriptome and metabolome have begun to provide information on the mechanisms by which optimum defence is achieved. However, it is clear that proper understanding of optimum defence cannot be gained through quantification of bulk changes at the whole plant or whole organ level, whether in global gene expression, or in bulk measures of defensive chemistry, such as total phenolics. What is required is more detailed temporal and spatial resolution of the responses of specific genes or compounds in the context of their function in the plant under biotic attack and different light conditions.

Whilst ecologists and molecular biologists have mostly taken different approaches to the question of interactions between light and defence, we feel that these approaches can provide an interface which can deliver benefits to both sets of disciplines. Work across these scales can be extremely effective in linking molecular responses with ‘real life’ ecological outcomes to stress (see, for example, work from the group of Ian Baldwin),

and we strongly encourage efforts to integrate molecular and ecological studies in all areas of biology.

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Table 1 Overview of field experiments into the effects of the light environment on (a) plant-herbivore interactions and (b) plant-pathogen interactions. These studies considered the effects of variation in total light, and in some cases responses have been attributed not just to photosynthetic radiation but the longer wavelengths of sunlight, resulting in changes in the thermal environment. The potential role of UV wavelengths was not considered in these studies. Key: +ve indicates that shade increases the leaf area eaten by a herbivore or infected by a pathogen, or has some beneficial effect on herbivore performance or behaviour (*e.g.* reduced mortality, increased growth rate, increased efficiency of food conversion etc.), -ve indicates negative responses, 0 indicates that shade treatments had no significant effect. na indicates not assessed.

Host/herbivore	Source of variation in the light environment	Effect of shading on leaf area eaten	Effect of shading on the herbivore	Source
<i>Fagus crenata</i> / Natural herbivore community	Natural variation with position in canopy	+ve	na	32
<i>Betula pubescens</i> / <i>Epirrita autumnata</i>	Natural variation with position in canopy	na	+ve	30
<i>Tilia cordata</i> / <i>Popillia japonica</i>	Natural variation with position in canopy	-ve	na	26
<i>Prunus mahaleb</i> / <i>Yponomeuta mahalebella</i>	Natural variation with position in canopy	na	-ve	1
<i>Nothofagus antarctica</i> and natural herbivore community	Natural variation with position in canopy	+ve	na	25
<i>Liriodendron tulipifera</i> and <i>Cornus florida</i> / Natural herbivore community	Range of natural field sites, plus artificial shading,	+ve	na	6
Five trees species / <i>Atta cephalotes</i>	Plants grown in full sun or partial shade	+ve	na	7
<i>Populus deltoides</i> / <i>Plagioderia versicolora</i>	“Open” versus “shade” sites	+ve	na	4
<i>Salix phylicifolia</i> / <i>Galerucella lineola</i> and <i>Salix myrsinifolia</i> / <i>Phratora vitellinae</i>	Field sites with or without tree canopy	+ve/ 0	+ve / 0	28
<i>Inga oerstediana</i> / <i>Atta cephalotes</i>	Understory, tree-fall gaps and full sun	-ve	na	21
<i>Cardamine cordifolia</i> / Natural herbivore community.	Removal of natural shade.	-ve	-ve	17
<i>Lycopersicon esculentum</i> / <i>Manduca sexta</i>	Artificial shading	+ve	na	11
<i>Betula pubescens</i> / <i>Epirrita autumnata</i>	Artificial shading	+ve	na	10
<i>Amaranthus palmeri</i> / <i>Spodoptera exigua</i>	Artificial shading	+ve	-ve/+ve	20
<i>Borrhichia frutescens</i> / <i>Pissonotus quadripustulatus</i>	Artificial shading	na	+ve	19
<i>Rhododendron mucronatum</i> / <i>Stephanitis pyrioides</i>	Artificial shading	-ve	+ve	3
<i>Vallisneria natans</i> / <i>Radix swinhoei</i>	midday fluxes 15-280 $\mu\text{mol m}^{-2} \text{s}^{-1}$	-ve	+ve/-ve	16

Table 1b The effects of the light environment on plant-pathogen interactions

Host/pathogen	Source of variation in the light environment	Effect of shading on infection	Source
Phlox / Erysiphe	Shaded or open sites in the field	+ve	12
<i>Anemome nemorosa</i> / <i>Tranzchelia anemones</i> <i>Anemome nemorosa</i> / <i>Ochropsora ariae</i>	Shaded or open sites in the field	+ve 0	8
<i>Agrostis stolonifera</i> / naturally occurring fungal pathogens	Shaded or open sites in the field	+ve	15
<i>Brachypodium sylvaticum</i> / <i>Epichloe sylvatica</i>	Shaded or open sites in the field	+ve	18
<i>Camellia sinensis</i> / <i>Exobasidium vexans</i>	Shaded or open sites in the field	+ve	19
<i>Camellia sinensis</i> / <i>Hemileia vastatrix</i>	Shaded or open sites in the field	-ve	29
<i>Quercus petraea</i> / <i>Microsphaera alphitoides</i>	Shaded or open sites in the field	-ve	13
<i>Betula papyrifera</i> and naturally occurring soil pathogens	Shaded or open sites in the field	+ve	
Forest tree seedlings/ <i>Pythium spp.</i>	Artificial shading	+ve	2
<i>Phacidium coniferarum</i>	Artificial shading	+ve	31
<i>Glycine soya</i> / <i>Sclerotinia sclerotiorum</i> <i>Medicago sativa</i> / <i>Verticillium albo-atrum</i> <i>Medicago sativa</i> / <i>Fusarium oxysporum</i>	Artificial shading	+ve +ve 0	24
<i>Euonymus fortunei</i> / <i>Colletotrichum gloeosporioides</i>	Artificial shading	-ve	22
<i>Picea mariana</i> / <i>Botrytis cinerea</i>	Artificial light treatments, pre-inoculation only	+ve	33
<i>Rhododendron sp</i> / <i>Erysiphe sp.</i>	Artificial light treatments, pre-inoculation only	+ve	14
<i>Triticum aestivum</i> / <i>Puccinia striiformis</i>	Artificial light treatments, pre-inoculation only	-ve	5
<i>Lycopersicon esculentum</i> / <i>Botrytis cinerea</i>	Artificial light treatments, pre-inoculation only	+ve	27

Literature cited in Table 1.

1, Alonso, 1997); 2, Augspurger & Kelly, 1984; 3, Bentz, 2003); 4, Crone & Jones, 1999); 5, de Vallavieille-Pope *et al.*, 2002; 6, Duds & Shure, 1994); 7, Folgarait *et al.*, 1996); 8, Garcia-Guzman & Wennstrom, 2001; 9, Gunasekera *et al.*, 1997; 10, Henriksson *et al.*, 2003); 11, Jansen & Stamp, 1997); 12, Jarosz & Levy, 1988; 13, Kelly, 2002; 14, Kenyon *et al.*, 2002; 15, Koh *et al.*, 2003; 16, Li *et al.*, 2005); 17, Louda & Rodman, 1996); 18, Meijer & Leuchtman, 2000; 19, Moon *et al.*, 2000); 20, Moran & Showler, 2005); 21, Nicholsons, 1991); 22, Ningen *et al.*, 2005; 23, O'Hanlon-Manners & Kotanen, 2004; 24, Pennypacker, 2000; 25, Rousseaux *et al.*, 2004; 26, Rowe & Potter, 1996); 27, Shafia *et al.*, 2001; 28, Sipura & Tahvanainen, 2000; 29, Soto-Pinto *et al.*, 2002; 30, Suomela *et al.*, 1995; 31, Wainhouse *et al.*, 1998; 32, Yamasaki & Kikuzawa, 2003; 33, Zhang *et al.*, 1995

Table 2 Overview of the effects of ultraviolet radiation on plant-herbivore interactions. These studies specifically manipulated ultraviolet radiation using lamps or wavelength-selective filters. Unless otherwise stated only UV-B (290-320nm) has been experimentally manipulated.

Host / herbivore	Experimental conditions	Effect of UV manipulation	Source
<i>Ipomoea batata</i> / <i>Bemisia tabaci</i> , <i>Frankliniella occidentalis</i> , or <i>Aphis gossypii</i> .	Polythene tunnels with ambient or attenuated total solar UV	Substantial reductions in attack by all three insects	1
<i>Zea mays</i> / <i>Ostrinia nubilalis</i>	+ or – UV in the glasshouse	Larvae preferred leaves grown without UV-B	2
<i>Oryza sativa</i> / <i>Helicoverpa armigera</i>	Artificial UV-B irradiation	Extracts of irradiated leaves had antifeedant, growth-inhibitory and antibiotic properties against larvae, and effects persisted into adults, which laid fewer, less viable eggs.	3
<i>Bemisia argentifolii</i> and <i>Frankliniella occidentalis</i>	Polythene tunnels with ambient or attenuated total solar UV	Insects dispersed preferentially into ambient UV environments, but UV had no effect on flight ability.	4
<i>Pisum sativum</i> / <i>Autographa gamma</i>	CE room with a range of UV-B doses	Increased UV-B increased leaf nitrogen and when foliage was fed to larvae this was correlated with an increase in larval growth rate and a reduction in the amount of plant material consumed.	5
<i>Trifolium repens</i> / <i>Spodoptera litura</i> or <i>Graphania mutans</i>	CE room with and without UV-B	36% reduction in weight of <i>S. litura</i> on foliage grown at high UV, but this depended on host genotype. <i>G. mutans</i> showed little response	6
<i>Glycine max</i> / <i>Caliothrips phaseoli</i>	Ambient or near zero UV-B in the field	UV-B reduced thrip herbivory: insects preferred leaves from reduced UV-B and avoided solar UV.	7
<i>Caliothrips phaseoli</i>	Ambient or near zero UV-B in the field	Insects preferred low UV-B environment	8
<i>Lolium perenne</i> and <i>Festuca spp.</i> / <i>Schistocerca gregaria</i>	Ambient and elevated UVA or UV-B in the field	No herbivore responses to excised leaves from different UV-B treatments except in <i>F. pratensis</i> where responses varied with UV treatment and/or endophyte infection of the host	9
<i>Plantago lanceolata</i> / <i>Precis coenia</i> or <i>Trichoplusia ni</i>	CE room at high ambient or above.	Growth of <i>T. ni</i> larvae was faster when fed excised leaves from elevated UV-B. Direct exposure of larvae to the UV treatments increased mortality of <i>T. ni</i> . UV had no significant effects on <i>P. coenia</i> .	10
<i>Trialeurodes vaporariorum</i>	Polythene tunnels with ambient or attenuated total solar UV	Attenuation of UV reduced whitefly dispersion, resulting in reduced populations in low UV tunnels	11
<i>Quercus robur</i> / natural herbivore community	Ambient and elevated UVA or UV-B in the field	Plants under elevated UV-B or UV-A suffered greater herbivory	12
<i>Gunnera magellanica</i> and natural herbivore community	Ambient or near zero UV-B in the field	Leaf area damaged increased under reduced UV-B.	13
<i>Gunnera magellanica</i> and natural herbivore community	Ambient or near zero UV-B in the field	Leaf area consumed increased 25-75% under attenuated UV-B	14

<i>Nothofagus antarctica</i> and natural herbivore community	Ambient or near zero UV-B in the field, and sun-exposed and shaded branches	Solar UV-B reduced insect damage by at least 30%, and this occurred with foliage in both sunny and shaded positions.	15
<i>Calluna vulgaris</i> / <i>Strophingia ericae</i> (Homoptera)	Ambient and elevated UV-B in the field	Increased UV-B reduced herbivore population density over two seasons	16
<i>Salix myrsinifolia</i> and <i>S. phylicifolia</i> / <i>Phratora vitellinae</i> or natural herbivore community	Ambient and elevated UV-B in the field	Herbivores more abundant under elevated UV-B but host did not suffer greater herbivore damage. Excised leaves of <i>S. phylicifolia</i> , from elevated UV-B reduced growth of <i>P. vitellinae</i> larvae compared with control leaves, but there was no comparable effect with leaves of <i>S. myrsinifolia</i> .	17
<i>Populus trichocarpa</i> / <i>Chrysomela scripta</i>	Zero, ambient and 2x ambient	Leaves from highest UV-B significantly reduced larval consumption efficiency	18
6 plant species and <i>Deroceras reticulatum</i> (Mollusca)	Ambient or near zero UV-B in the field	Significant effects in two of the six species. In <i>Nothofagus antarctica</i> , leaf area consumed reduced by 2/3rds in foliage from under near-ambient UV-B. In <i>Carex decidua</i> twice as much as leaf area was consumed in reduced UV-B radiation.	19
<i>Glycine max</i> / <i>Anticarsia emmatalis</i> or natural herbivore community	Ambient or near zero UV-B in the field	Leaves from reduced UV-B were more attractive to larvae, supported higher growth rates and lower mortality. No direct effect of UV exposure on larval mortality. Attenuation of UV increased natural herbivore damage by 2-fold.	20
<i>Morus nigra</i> / <i>Bombyx mori</i>	Artificial UV irradiation in CE rooms	UV treatments reduced consumption of foliage by larvae.	21

Literature cited in Table 2.

1, Antignus *et al.*, 1996; 2, Bergvinson *et al.*, 1995; 3, Caasi-Lit, 2005; 4, Costa & Robb, 1999; 5, Hatcher & Paul, 1994; 6, Lindroth *et al.*, 2000; 7, Mazza *et al.*, 1999; 8, Mazza *et al.*, 2002; 9, McLeod *et al.*, 2001; 10, McCloud & Berenbaum, 1999; 11, Mutwiwa *et al.*, 2005; 12, Newsham *et al.*, 1999; 13, Rousseaux *et al.*, 1998; 14, Rousseaux *et al.*, 2001; 15, Rousseaux *et al.*, 2004; 16, Salt *et al.*, 1998; 17, Veteli *et al.*, 2003; 18, Warren *et al.*, 2002; 19, Zaller *et al.*, 2003; 20, Zavala *et al.*, 2001; 21, Yazawa *et al.*, 1992.

Figure Legends:

Figure 1. Impacts of light on plant resistance against pests and pathogens.

Different forms of biotic attack (top row) activate different major routes to resistance (second row), as well as repair and healing mechanisms. Light can act positively (solid arrows) or negatively (barred lines), via a number of distinct pathways. Many of these affect the generation of reactive oxygen species, which appears to be a key node for the interactions between light and defence.

