

The shade avoidance syndrome: multiple responses mediated by multiple phytochromes

H. SMITH & G. C. WHITELAM

Department of Botany, University of Leicester, Leicester LE1 7RH, UK

ABSTRACT

In recent years, the concept of shade avoidance has provided a functional meaning to the role of the phytochrome photoreceptor family in mature plants in their natural environment, and the question of which of these phytochromes is responsible for shade avoidance reactions has inevitably been raised. Unfortunately, a misconception has arisen that phytochrome B is solely responsible for detecting the environmental signal that initiates the shade avoidance syndrome. This view is too simplistic, and is based upon a selective interpretation of the available evidence. In this short Commentary, we review the concept of the shade avoidance syndrome, show how the misconception arose, and emphasize the plurality of perception and response that is crucial to successful competition for light.

Key-words: mutants; phytochromes; shade avoidance.

THE SHADE AVOIDANCE SYNDROME

Whenever plants grow in close proximity, in forests, in herbaceous communities, in grassland swards or in hedgerows, there is competition for light. The resource of radiant energy in dense plant stands is unreliable and patchy, and evolution has provided plants with two principal approaches to provide for survival under such environmental conditions. Essentially, plants may avoid shade, or they may tolerate shade. The angiosperms in particular have evolved impressive capacity to avoid shade, and this may be one of the factors that have contributed to their success. Shade avoidance represents one of the most important competitive strategies that plants possess, and its effectiveness is undoubtedly a consequence of the multiplicity of responses that are available to the shaded plant. Responses to shade are many and varied, and it is now fully accepted that shade avoidance reactions are all initiated by a single environmental signal, the reduction in the ratio of red (R) to far-red (FR) radiation (i.e. R:FR) that occurs within crowded plant communities. We use the term 'syndrome' to describe the multiple responses to low R:FR, in analogy to medical conditions in which multiple symptoms are caused by a single underlying problem.

The concept of shade avoidance has been with us for at least 20 years, although tracing the origin of the term is

somewhat difficult. In the first half of this century, there was a great deal of research on the responses of plants to artificial shade, using neutral density screens to simulate the reduction in irradiance that occurs in natural plant canopies. That research must now be regarded as essentially irrelevant, as the reduction in irradiance under shade is now known not to be a reliable signal. The earliest reported observations linking phytochromes to shade avoidance responses are probably those of Cumming (1963), who demonstrated that the germination of *Chenopodium rubrum* seeds was sensitive to R:FR over a wide range, and speculated that this behaviour may be important in optimizing germination in relation to the presence of vegetation shade. At about the same time, the pioneers of modern photomorphogenesis, Hendricks & Borthwick (1963), remarked, almost in passing, that overhanging foliage might modify vegetative development through effects on stem and leaf growth. Kasperbauer and colleagues, in a number of publications, noted the importance of FR light filtered through or reflected by vegetation in crop plants, particularly in relation to the orientation of planting rows (e.g. Kasperbauer 1971).

The demonstration that shade avoidance reactions are phytochrome-mediated via the perception of the relative amounts of R and FR radiation came as a result of quantitative measurements and simulation experiments carried out in the 1970s. First, natural radiation spectra were analysed and summarized in terms of R:FR ratio (Holmes & Smith 1975, 1977). These natural variations were then related to estimated Pfr/P, the phytochrome photoequilibrium, the relationship being a rectangular hyperbola (Smith & Holmes 1977). By simulating shade avoidance extension growth responses using artificial light sources which provided uniform photosynthetically active radiation (PAR) but which varied in R:FR, the role of phytochrome-perceived variations in light quality was then firmly established (Morgan & Smith 1976, 1978, 1981). The range of responses to reduced R:FR ratio correlated identically with the observed growth responses of plants to shade in the natural environment, and indeed plants naturally adapted to shade conditions showed weaker responses to R:FR than did those adapted to open conditions (Morgan & Smith 1979).

Ecologists have long been used to the idea that plants avoid shade, and Grime (1979), in his book on vegetation strategies, used the term 'shade avoidance' as an index term, although it is difficult to find the term in the text! In

Correspondence: Harry Smith, Department of Botany, University of Leicester, Leicester LE1 7RH, UK.

the natural environment, aggressive shade-avoiding species exhibit strong elongation responses in shade, summarized by Grime (1979) as follows: 'in response to shade plants produce less dry matter, retain photosynthate in the shoot at the expense of root growth, develop longer internodes and petioles, and produce larger thinner leaves'. The adaptive significance of shade avoidance has recently been demonstrated in relation to the adaptive plasticity concept (Schmitt *et al.* 1995) and is discussed in detail by Schmitt (1997). The ecological significance of shade avoidance is reviewed by Ballaré *et al.* (1997).

When vegetation shade is simulated in growth cabinets in which R:FR is low but PAR sufficient to allow for sustained growth, these phenological changes are exaggerated. Table 1 shows the main categories of response that are observed in plants growing under simulated shade conditions. It can be seen that shade avoidance responses are important throughout the whole life cycle, from germination to flowering and seed set. Germination under dense canopies would clearly be disadvantageous for seeds with small reserves; phytochrome-mediated shade avoidance responses are evident at this stage with low R:FR inhibiting germination and imposing secondary dormancy. In some cases, notably those of pioneer trees, germination of seed held dormant in the soil bank requires a substantial daily period of high R:FR radiation, such as only occurs in large canopy gaps (Vasquez-Yanes & Smith 1982). Thus, shade avoidance responses

allow for optimum germination appropriate to environmental conditions.

The most dramatic shade avoidance response, seen both in natural shade and in low R:FR simulations, is the stimulation of elongation growth. This response may not only be quantitatively large, it can also be remarkably rapid, with lag phases of a few minutes in some cases (Child & Smith 1987). In simulation experiments, extreme responses can be obtained when the photosynthetically active radiation is maintained at reasonable levels, allowing the provision of sufficient resources for shade avoidance to be maximized. In our laboratory, in a 3 week experiment, we have grown sunflowers to 1 m tall under low R:FR radiation, when the controls grown in high R:FR reached only 25 cm! Elongation responses to low R:FR are most easily observed in internodes, but petioles also show strong responses. In the monocots, elongation of leaves, and of leaf sheathes, is stimulated by low R:FR. Tendrils and other organs capable of polar longitudinal growth all show responses to low R:FR. Concomitant with stem elongation (in dicots) is often a reduction in leaf development, although this can be variable. In some species, but not all, leaf area growth is reduced under low R:FR. A more general response is a reduction in leaf thickness, and in some cases a complete breakdown of the characteristic palisade and spongy mesophyll anatomy is observed (McLaren & Smith 1978). Other aspects of leaf development are also modified during shade avoidance including, commonly, a substantial reduction in chlorophyll production, readily observed by the naked eye. More variable are changes in the ratio of chlorophylls *a:b*, which is sometimes reduced and sometimes elevated under shade conditions. Essentially, however, shade avoidance responses result in increased shoot extension at the expense of leaf development. This is manifested as a marked strengthening of apical dominance and reduction in branching in dicots, or tillering in grasses (Casal *et al.* 1986). Associated with increased apical dominance is a commonly seen phenomenon in which leaf angle is increased in response to low R:FR; in other words, leaves tend to re-orientate upwards under simulated shade conditions (Whitelam & Johnson 1982).

A very important component of the shade avoidance syndrome is an acceleration of flowering, seen clearly in *Arabidopsis* (Halliday *et al.* 1994), but readily observable in all shade-avoiding plants. Although the adaptive significance of this response to impending shade has not been adequately investigated, it could reasonably be argued that accelerated flowering and seed production under shade increase the probability of the survival of the organism, and therefore of the species. Accelerated flowering under low R:FR is associated with reduced seed set, truncated fruit development and often a severe reduction in the germinability of the seed produced. Overall, shade avoidance involves a marked redirection of assimilates towards elongation and away from structures dedicated to resource acquisition and storage.

All of the responses collected together here under the shade avoidance syndrome are observable in natural, dense

Table 1. The shade avoidance syndrome

Physiological process	Response to shade (i.e. reduced R:FR ratio)
Germination	Retarded
Extension growth	Accelerated
Internode extension	Rapidly increased (lag c. 5 min)
Petiole extension	Rapidly increased
Leaf extension	Increased in cereals
Leaf development	Retarded
Leaf area growth	Marginally reduced
Leaf thickness	Reduced
Chloroplast development	Retarded
Chlorophyll synthesis	Reduced
Chlorophyll <i>a:b</i> ratio	Balance changed
Apical dominance	Strengthened
Branching	Inhibited
Tillering (in cereals and grasses)	Inhibited
Flowering	Accelerated
Rate of flowering	Markedly increased
Seed set	Severe reduction
Fruit development	Truncated
Assimilate distribution	Marked change
Storage organ deposition	Severe reduction

communities, and can be simulated by growing plants under low R:FR ratio conditions. Furthermore, by simply exposing plants to horizontal FR radiation with white light from above, similar responses are induced, consistent with the notion that plants anticipate impending shading by detecting FR reflection signals from neighbouring vegetation (Morgan & Smith 1981; Child & Smith 1987; Ballaré *et al.* 1987). The question therefore becomes: which phytochromes are responsible for sensing FR reflection signals and for mediating the shade avoidance syndrome?

HOW DID THE ASSUMPTION THAT *phyB* IS SOLELY RESPONSIBLE FOR MEDIATING SHADE AVOIDANCE RESPONSES ARISE?

The long hypocotyl (*lh*) mutant of cucumber was one of the first mutants deficient in phytochrome B (*phyB*) to be characterized in any detail. Spectrophotometric and immunochemical analyses of the phytochrome status of etiolated and light-grown *lh* plants provided evidence that, whilst the mutant possessed wild-type levels of light-labile phytochrome A (*phyA*), it showed a deficiency in the light-stable phytochrome pool; specifically, a polypeptide species reactive with a monoclonal antibody raised against a recombinant fragment of tobacco PHYB was absent in extracts of *lh* seedlings (Adamse *et al.* 1988; López-Juez *et al.* 1992). Prior to the demonstration that *lh* lacks an immunochemically detectable PHYB-like protein, it was established that seedlings of the *lh* mutant had aberrant responses to light (e.g. Adamse *et al.* 1987) and that light-grown *lh* seedlings resemble wild-type seedlings showing the shade avoidance syndrome (e.g. López-Juez *et al.* 1990; Ballaré *et al.* 1991). Moreover, it was reported that already elongated *lh* seedlings show no further elongation responses to end-of-day (EOD) FR light treatments or to supplementary FR during the photoperiod (e.g. Adamse *et al.* 1988; López-Juez *et al.* 1990; Ballaré *et al.* 1991). From these observations it was concluded that *lh* seedlings were completely devoid of the photoresponses mediated by the phytochrome(s) that was active in shade detection. Since *lh* seedlings were subsequently shown to lack a PHYB-like polypeptide (López-Juez *et al.* 1992), it is inferred that *phyB* (alone) mediates responses to vegetational shade in cucumber.

The analysis of *phyB*-deficient mutants in other species, most notably the *phyB*-null mutants of *Arabidopsis*, confirmed the striking similarity between the phenotypes of such mutants and the phenotypes of wild-type plants displaying the shade avoidance syndrome (e.g. Nagatani *et al.* 1991; Somers *et al.* 1991; Devlin *et al.* 1992; Reed *et al.* 1993). This, too, lent support to the notion that *phyB* mediates responses to vegetational shade.

EVIDENCE FROM MUTANT PLANTS THAT OTHER PHYTOCHROMES ARE INVOLVED

Despite initial suggestions that *phyB*-deficient mutants showed no responses to EOD FR or to supplementary FR

during the photoperiod, it is now apparent that many such responses are detectable in this class of mutants. For instance, the hypocotyls of light-grown cucumber *lh* seedlings, although already elongated, show a significant additional elongation response to supplementary FR (Whitelam & Smith 1991; Smith *et al.* 1992). This response is a classical element of the shade avoidance syndrome. These findings could indicate that the *lh* mutation is leaky, and so produced some functional *phyB*, or they could indicate that phytochromes other than the *phyB*-like species that are absent in *lh* are also able to mediate responses to the R:FR ratio.

Null alleles of the *Arabidopsis phyB* mutant also show typical shade avoidance responses to supplementary FR given during the photoperiod and to EOD FR treatments (e.g. Whitelam & Smith 1991; Goto *et al.* 1991; Robson *et al.* 1993; Halliday *et al.* 1994; Devlin *et al.* 1996). Both daytime reduction in R:FR ratio and EOD FR treatments induce an early flowering response in wild-type *Arabidopsis* seedlings. This represents an obvious manifestation of the shade avoidance syndrome in many plants. Although *phyB*-null mutants are early flowering under control conditions, they nevertheless display a clear early-flowering response to simulated vegetational shade (Whitelam & Smith 1991; Goto *et al.* 1991; Halliday *et al.* 1994; Devlin *et al.* 1996). *Arabidopsis* mutants that are null for *phyB*, although already elongated, also show increased elongation growth responses to both reduced R:FR ratio and EOD FR (Devlin *et al.* 1996). These observations provide a very clear indication that *phyB* is not the sole mediator of the shade avoidance syndrome in *Arabidopsis*.

The phenotype of the *Arabidopsis phyB* mutant is rather variable and does not always phenocopy wild-type plant responses to low R:FR ratio. Thus, whereas low R:FR ratio always leads to a decrease in leaf area and a decrease in specific stem weight in wild-type seedlings, the *phyB* mutant can sometimes constitutively display increased leaf area and increased specific stem weight (Robson *et al.* 1993). Furthermore, since leaf area and specific stem weight of the *phyB* mutant respond to low R:FR ratio in the same way as in wild type, these shade avoidance responses of the *phyB* mutant are sometimes exaggerated (Robson *et al.* 1993).

Through the analysis of *phyA* mutants, and *phyA phyB* double mutants, it is apparent that *phyA* is not necessary for display of the shade avoidance syndrome in *Arabidopsis* (Yanovsky *et al.* 1995; Devlin *et al.* 1996; Whitelam & Devlin 1997). In fact, at least during seedling establishment, the action of *phyA* in plants exposed to low R:FR ratio antagonizes that of *phyB* in the control of elongation growth (Yanovsky *et al.* 1995; Smith *et al.* 1997). Consequently, *phyA* mutants display such exaggerated elongation responses to low R:FR ratio that many of them die. This suggests that a possible role for *phyA* in de-etiolating seedlings is to limit some of the shade avoidance responses.

Recently, the retained shade avoidance responses of *Arabidopsis phyA phyB* double mutants has been exploited

in screens to identify new photoreceptor mutants. Significantly, some mutants that show no detectable additional responses (flowering time and/or elongation growth) to either supplementary FR during the photoperiod or to EOD FR have been isolated (P. F. Devlin and G. C. Whitelam, unpublished results). The analysis of these mutants may provide information about the involvement of photoreceptors in the shade avoidance syndrome.

Analysis of the *tri* mutant of tomato (see Kendrick *et al.* 1997) provides compelling evidence that phyB is not the sole mediator of the shade avoidance syndrome in all plants. This mutant has been shown to be deficient in a homologue of phyB (van Tuinen *et al.* 1995; Kerckhoffs *et al.* 1996). However, unlike many other phyB-deficient mutants, light-grown *tri* seedlings do not obviously resemble the shade avoidance syndrome of wild-type plants. Furthermore, *tri* seedlings show more or less normal responses to both supplementary FR during the photoperiod and EOD FR (e.g. Kerckhoffs *et al.* 1992). The observation that phyB is not necessary for the shade avoidance syndrome in tomato is consistent with the notion that phyB does not play a significant role in these responses. A similar situation exists in *Nicotiana plumbaginifolia* in which two mutants have been isolated and characterized that have lesions in a *PHYB* orthologue, and are null for the phyB photoreceptor (M. Hudson, P. R. H. Robson, Y. Kraepiel, M. Caboche and H. Smith, unpublished results). These mutants have normal responses to low R:FR ratio. However, the possibility that there is redundancy among the phytochromes of tomato and *N. plumbaginifolia* with respect to the shade avoidance syndrome cannot be dismissed.

CONCLUSIONS

Despite initial attempts to ascribe the shade avoidance syndrome to the action of a single member of the phytochrome family, it is now clear that multiple phytochromes are involved. This is perhaps not surprising given the complexity and importance of these responses. Furthermore, it seems likely that the contributions of different members of the phytochrome family to the shade avoidance syndrome, and the degree of redundancy among the phytochromes, will be different in different plant species. Thus, conclusions drawn from the analysis of one plant species cannot be universally applied.

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