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The genetics of phytochrome signalling in Arabidopsis

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The application of Arabidopsis genetics to research into the responses of plants to light has enabled rapid recent advances in this field. The plant photoreceptor phytochrome mediates well-defined responses that can be exploited to provide elegant and specific genetic screens. By this means, not only have mutants affecting the phytochromes themselves been isolated, but also mutants affecting the transduction of phytochrome signals. The genes involved in these processes have now begun to be characterized by using this genetic approach to isolate signal transduction components. Most of the components characterized so far are capable of being translocated to the cell nucleus, and they may help to define a new system of regulation of gene expression. This review summarises the ongoing contribution made by genetics to our understanding of light perception and signal transduction by the phytochrome system.

Key words: photoreceptors / phytochrome / signal transduction / pathway / mutagenesis / *Arabidopsis thaliana*

Introduction

Plants are extremely sensitive to small changes in environmental conditions, and plant physiologists are concerned with the mechanisms of these responses. Some of the most important and best studied of these responses are the responses to light. Plants are particularly sensitive to light in the red and far-red regions of the visible spectrum, and a family of photoreceptors called phytochromes mediates their responses to these wavelengths of light. The phytochromes are chromoproteins that exist in two stable forms, Pr and Pfr, which are interconverted by light. These unique properties of phytochromes give it them characteristic photoreversible spectral signature in red and far-red light. There are currently five known phytochrome species in Arabidopsis, and most higher plants are thought to have three or more,[1] all of which require a common chromophore, phytochromobilin. In conjuction with the blue and ultraviolet light photoreceptors, phytochromes give plants their extraordinary sensitivity and range of responses to their principal energy source, the light environment.[2]

Sage[3] has written an excellent history of the field, including details of all the classic experiments. However, since the beginning of the 1990s, the phytochrome field has been invaded by mutants. Recent reviews have provided extensive overviews of phytochrome mutant genetics.[4–7] Since more is known about the genetics of this system in Arabidopsis than in all other plant species, this review will be restricted to this model species. It is the purpose of this review to discuss the evidence gained from genetic approaches to the study of the perception of light signals by plants, and to attempt to integrate this limited knowledge with that gained from other approaches.

Photomorphogenic mutants

The study of mutations that affect the responses of plants to light began with photoperiodic mutants.[3] Phytochromes have been shown to be important in the perception of photoperiod and the determination of flowering time by many.[3,8,9] For this reason, the processes of phytochrome signalling may involve some of the many mutants and genes known to affect the timing of flowering. This is a field which has been reviewed elsewhere,[10] but it is worth remembering how closely tied this field is with that of phytochrome signalling.

After the flowering mutants, the next class of photomorphogenic mutants to be discovered was that of

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the loci involved in de-etiolation. These fall into two classes: those that produce light-dependent phenotypes, and those that cause seedling morphogenesis to follow the photomorphogenic path even in complete darkness. The first class is likely to consist of mutants compromised in the processes of light perception and signalling, and the typical phenotypes of this class are shown in Figure 1. The most obvious characteristic of this class of mutants is altered hypocotyl elongation in light-grown plants.[11] The second class, the *cop*, *det* and some *fus* mutants, have short hypocotyls and photomorphogenic phenotypes as dark grown seedlings, and have been reviewed elsewhere.[12–15] The fact that mutations in many of these loci produce highly pleiotropic phenotypes makes their role unlikely to be restricted to light signalling. Rather, they are likely to control multiple processes of cellular development, as evidenced by the presence of *COP* genes in animals.[16] The systems that they define in plants are certainly light regulated, but probably act downstream of photoreceptor-specific components in phytochrome signalling pathways.[6] (See article by Schwechheimer and Deng, this volume.)

The mutants that show the most obvious deficiency in light-induced responses are those that affect synthesis of the phytochrome chromophore. The phenotypes of the chromophore mutants and the phytochrome photoreceptor mutants are summarized in Figure 1 and Table 1. Different genes of the *PHY* gene family encode the protein components of the different phytochrome species. Hence, a mutant in a single phytochrome gene does not lose all its responses to red or far red light, due to redundancy within the gene family. In contrast, since phytochrome requires covalently attached phytochromobilin to detect light, mutations in the enzymes of bilin synthesis may affect all the phytochromes in the plant. These mutants can be shown to lack the spectral signature of phytochrome and show etiolated phenotypes throughout their development, whatever the light environment.[17, 18] The two Arabidopsis chromophore deficient mutants, *hy1* and *hy2*, have the most strikingly etiolated phenotype of all the known Arabidopsis photomorphogenic single mutants.[11] The *hy1* mutant locus is now cloned; HY1 encodes a bilin synthetic enzyme, a haem oxygenase.[19, 20]

Mutations in the genes encoding photoreceptors can also produce strong phenotypes, especially under certain light conditions or developmental stages (Figure 1, Table 1). This gives us a strong clue as to which of the many phytochromes are the most important for perception of a given light stimulus.[21] Mutations disrupting the *PHYTOCHROME B (PHYB)* gene are known in many species, and so far invariably produce a plant which is elongated relative to the wild type when grown under white or red

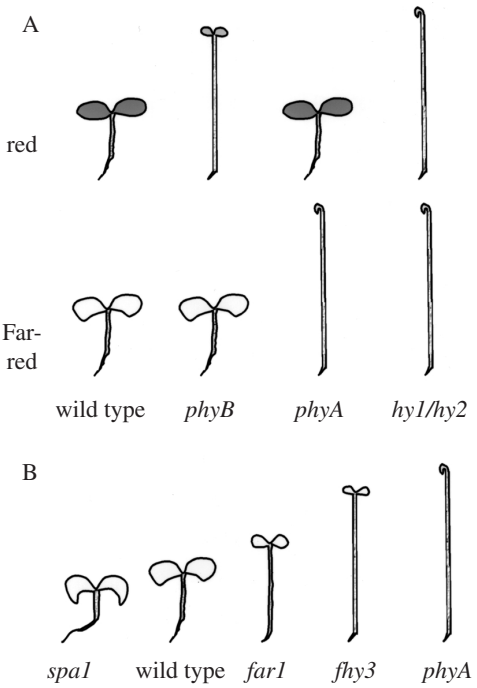

Figure 1. A Expected phenotype of wild type and major photoreceptor mutants of Arabidopsis, as seen after treatment with red or far-red light for 3 days after germination. B Expected phenotype of wild-type, *phyA* mutant and examples of different types of phyA-specific signalling mutants as they would be seen after treatment with far-red light for 3 days after germination. The mutations shown cause increases or decreases in the sensitivity of phyA responses.

light.[17, 22, 23] The *phyB* mutants are also deficient in the shade-avoidance syndrome, implying responses mediated by this phytochrome can control the later development of light-grown plants.[2, 24] Null mutations in the *PHYA* gene produce a negligible phenotype under 'normal' (i.e. white light) growth conditions.[25] However, these *phyA* mutations eliminate the normally strong response of seedlings to far-red light given alone.[26, 27] Mutants in the other members of the phytochrome gene family, *PHYC*, *PHYD* and *PHYE*, have proved more elusive. A natural *phyD* mutant is now known.[28] This null mutation was present in an Arabidopsis ecotype (Ws) isolated from the wild, illustrating that the minor role for which phyD is required is one that Arabidopsis can readily live without. A *phyE* mutant has been isolated by means of a screen that took into account potential redundancy with other phytochromes by using a background line lacking phyA and phyB.[29] When outcrossed from the mutant background in which it was isolated, however, this mutant shows no discernible phenotype. A *phyC* mutant remains elusive at the time of writing, perhaps because the phenotype,

Table 1. Phytochrome photoreceptor mutants of Arabidopsis. Earlier or alternative names for mutants are given in parentheses

Mutant	Phenotype	Type of mutation	Cloned?	Reference
hy1	loss of red and far-red induced de-etiolation, pale, elongated	chromophore biosynthesis	haem oxygenase	Koornneef *et al.*, 1980,[11] Muramoto *et al.*, 1999,[19] Davis *et al.*, 1999[20]
hy2	loss of red and far-red induced de-etiolation, pale, elongated	chromophore biosynthesis	unknown as yet, likely to be phytochromobilin synthetic enzyme	Koornneef *et al.*, 1980,[11] Terry, 1997[18]
phyA (fhy2) (hy8) (fre1)	total loss of far-red induced de-etiolation	apoprotein	many alleles, lesions in *PHYA* gene	Whitelam *et al.*, 1993,[25] Parks and Quali, 1993,[26] Nagatani *et al.*, 1993[27]
phyB (hy3)	loss of red induced de-etiolation, early flowering, elongated	apoprotein	many alleles, lesions in *PHYB* gene	Koornneef *et al.*, 1980,[11] Reed *et al.*, 1993[22]
phyD	slightly reduced red light responses (stronger, elongated phenotype in absence of phyB)	apoprotein	one lesion known in *PHYD* gene, natural mutation in WS ecotype	Aukerman *et al.*, 1997,[28] Devlin *et al.*, 1999[30]
phyE	no monogenic phenotype; elongated, early flowering in absence of phyA and phyB	apoprotein	one lesion known in *PHYE* gene	Devlin *et al.*, 1998[29]

if present, will be even more difficult to detect. It seems likely, therefore, that in a wild-type plant phytochromes A and B mediate the bulk of the developmental responses of plants to light in the red/far red region of the spectrum. The exact role for which selective pressure exists for phyD and E to be retained is debatable; most of their functions are only visible in the *phyA phyB* double mutant background.[9, 28–30] The redundancy they provide may be the key to their retention, rather than any specific response for which they are required in wild-type Arabidopsis.

Signal transduction mutants

The characterization of the phytochrome photoreceptors, their chromophores and null mutants, has defined specific response pathways from these receptors. Interest has now moved on to elucidating the mechanisms of these signalling pathways, downstream of the phytochromes themselves. Since the phenotype of the phytochrome mutants is known, screens for phytochrome-signal transduction mutants are now easy to design. Such mutants are characterized by decreased or increased responses to specific light conditions (Figure 1). Several such mutants are now known, and are discussed below.

General light signalling mutants

The constitutively de-etiolated mutants, such as the *cop* and *det* mutants discussed earlier, are probably affected in the later stages of light signalling.[12, 13] There are also mutants that show a general reduction in all types of light signalling. The best characterized of these is *hy5*, which is now known to be mutated in a gene encoding a b-ZIP transcription factor.[31] The action of HY5 is thought to be repressed by COP1[32] which induces degradation of HY5.[33] As a component involved in blue, red and far-red induced responses, along with basic processes of plant development, HY5 is currently thought to be downstream of convergence points between signals from multiple photoreceptors and other developmental signals.

The *shy2* mutant was originally isolated as a suppressor of *phyB* mutants and of phytochrome chromophore mutants.[34, 35] The mutation has been cloned and found to correspond to IAA3, one of the early-auxin-inducible genes.[36] Another *phyB* suppressor mutant is *bas1*, which is affected in brassinosteroid metabolism.[37] While specific roles for these components in early steps of phytochrome signalling are not suggested by this work, these genes and others like them may provide a downstream out-

put for light signalling, via growth regulation, to morphogenesis. Two more mutants affecting responses from multiple photoreceptors are *pef1*[38] and *psi2*.[39] The *pef1* mutant shows attenuated red and far-red responses; hence it is phytochrome specific, but not specific to a single receptor. The *psi2* mutant is hypersensitive to red and far-red light, and has necrotic lesions in light-grown plants (a phenotype which is suppressed in the presence of the *phyB* mutation). The *PSI2* gene product may therefore play a role in a hitherto uncharacterized mechanism of light-controlled development involving cell death.

Photoreceptor specific mutants

For the reason that mutants specifically affecting signalling from a single photoreceptor may be more likely to be blocked in early, light specific steps, many investigators have concentrated on such mutants. Many such specific light signalling mutants are now known, and a summary of them is given in Tables 2 and 3.

Phytochrome A signalling

The mutant screen that first allowed the isolation of *phytochrome A (phyA)* mutants in Arabidopsis was the growth of seedlings under far-red light alone. Wild-type Arabidopsis seedlings respond strongly to light of this wavelength, in two distinct response modes; the very low fluence response (VLFR), and the high irradiance response (HIR).[2] The most obvious effect of the HIR response is a marked reduction in hypocotyl elongation, which would otherwise occur in dark-grown seedlings. Consequently, mutants deficient in the perception of far-red light are easily screened for, as they are much taller than wild-type seedlings under these conditions (Figure 1). The *phyA* mutants completely lack this response, which is solely mediated by phyA. Using the same screens in which the *phyA* mutant was isolated, other mutants were detected that were reduced in their sensitivity to far-red (see Table 2). The extent to which these responses are lost varies between the different mutants, some being strongly affected and others only weakly.

The far-red response mutants *fhy1* and *fhy3*[25] were the first to be isolated, and still have the strongest known phenotypes, with the possible exception of *fin2*.[40] They have a phenotype consistent with a severe reduction in the efficiency of phyA signalling, but are not completely blind to far-red light alone, as are null mutants in the *phyA* gene itself. The loci mutated in these plants are consequently good candidates for genes encoding important components of a phyA-specific signalling pathway. The components they encode are likely to be upstream of any branch point in a general phytochrome signalling pathway or network (Figure 2). No sequence of the genes involved was available at the time of writing.

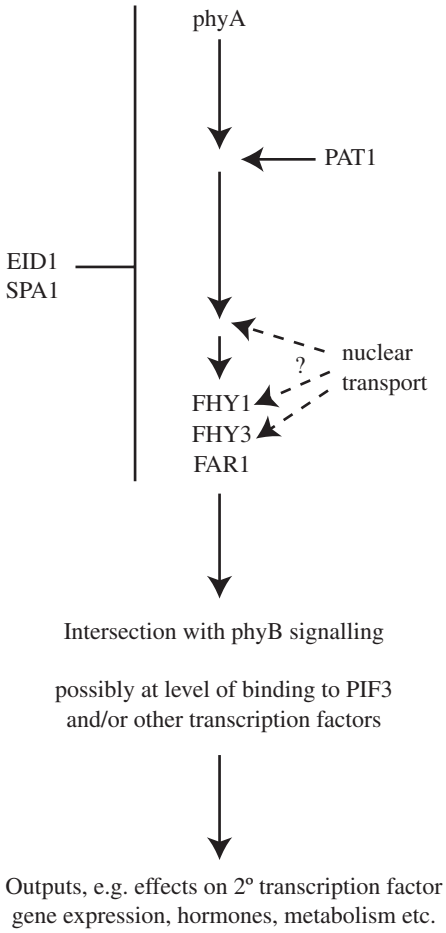

Figure 2. Highly speculative order of components in phyA signal pathway, deduced from subcellular localization and specificity to the photoreceptor.

The most intriguing aspect of the *fhy3* mutant is that it shows qualitatively normal responses to red light, and a wild-type phenotype as a light-grown adult.[25,41] The only response that *fhy3* clearly affects is the HIR; the VLFR, also mediated by phyA, seems to be intact. Mutations at the *fhy3* locus are therefore thought specifically to block the HIR pathway.[41] The *fhy1* mutant is also specific to phyA, but affects both the HIR and the VLFR.[41]

An intriguing new phyA signalling mutant is *pat1* (**p**hytochrome **A** signal **t**ransduction).[42] The phenotype is similar to that of *fhy1* and *fhy3*, i.e. a strong reduction in sensitivity to far-red light. Like previously characterized far-red response mutants, the phenotype appears to affect

Table 2. Putative phytochrome A early-signal transduction mutants of Arabidopsis

Mutant	Phenotype	Specificity	Cloned?	Reference
fhy1 *fhy3* *fin2*	loss of far-red induced de-etiolation	phyA	not yet	Whitelam *et al.*, 1993,[25] Soh *et al.*, 1998[40]
pat1	loss of far-red induced de-etiolation	phyA	negative-dominant truncation in cytoplasmic VHIID/GRAS protein.	Bolle *et al.*, 2000[42]
far1	partial loss of far-red induced de-etiolation	phyA	null mutation in nuclear protein with no obvious functional motifs	Hudson *et al.*, 1999[46]
pef1	partial loss of red and far-red induced de-etiolation	multiple phytochromes	not yet	Ahmad *et al.*, 1996[38]
psi2	gain in sensitivity of red and far-red induced de-etiolation	multiple phytochromes	not yet	Genoud *et al.*, 1998[39]
eid1	gain in sensitivity of far-red induced de-etiolation	phyA*	not yet	Buche *et al.*, 2000[49]
spa1	gain in sensitivity of far-red induced de-etiolation	phyA*	WD repeat protein with kinase homology	Hoecker *et al.*, 1998,[47] 1999[48]

*Indicates specificity determined by epistasis of *phyA* photoreceptor mutation.

Table 3. Putative phytochrome B early-signal transduction mutants of Arabidopsis

Mutant	Phenotype	Specificity	Cloned?	Reference
red1 *pef2* *pef3*	partial loss of red induced de-etiolation	phyB	not yet	Wagner *et al.*, 1997,[51] Ahmad *et al.*, 1996[36]
pef1	partial loss of red and far-red induced de-etiolation	multiple phytochromes	not yet	Ahmad *et al.*, 1996[38]
poc1	gain in sensitivity of red induced de-etiolation	phyB*	promoter insertion in gene for PIF3; basic helix-loop-helix transcription factor	Halliday *et al.*, 1999[53]
psi2	gain in sensitivity of red and far-red induced de-etiolation	multiple phytochromes	not yet	Genoud *et al.*, 1998[39]

*Indicates specificity determined by epistasis of *phyB* photoreceptor mutation.

only processes directly attributable to phyA signalling. The mutation is the result of a T-DNA insertion in a gene encoding a so-called GRAS protein. There are strong precedents for this family of proteins being involved in plant signal transduction, such as the GAI[43] and SCARECROW[44] proteins.[45] The *pat1* mutation causes truncation of an open reading frame, presumably causing the production of a truncated protein. This mutant protein

appears to have a negative-dominant effect on phyA signalling. The phenotype is replicated by expression of the truncated gene in a wild-type background, and the mutant is partially complemented by overexpression of the wild-type gene. Unfortunately, although this is convincing evidence for involvement of this protein in phytochrome A signalling, it does not allow direct comparison with the EMS or gamma induced, presumably loss-of-function mutants such as *fhy3*, described above. Redundancy may again explain why null mutations at the *pat1* locus have not been previously isolated in far-red response screens. The most intriguing aspect of the PAT1 protein is that it appears to be the first phytochrome-signalling component known to be cytoplasmically localized. This carries an implication that it may potentially be involved in very early steps indeed in phytochrome A signal transduction; i.e. before phyA is translocated to the nucleus (Figure 2).

The *far1*[46] mutant has a weaker phenotype than the above. Isolated in a screen of mutagenized phyA over-expressing seedlings, the *far1* mutant shows a subtle but significant decrease in response to far-red light, both in the phyA-over-expressing and wild-type backgrounds. The positional cloning of *far1* demonstrated that the locus encodes a nuclear-localized protein with no substantial homology to proteins with previously characterized functions. However, strongly homologous genes exist in Arabidopsis and in other angiosperm species, including monocotyledons and dicotyledons. The presence of *far1*-like gene family may mean that redundancy is the reason for the incomplete block in phyA signalling in the far1 mutant, and the FAR1 family of proteins may perform a necessary step in this pathway.

The reverse phenotype to *fhy1*, *far1* etc is displayed by *spa1*,[47] a suppressor mutant of a weak allele of *phyA*.[47] When in a wild-type genetic background, the *spa1* mutation causes plants to be hypersensitive to both red and far-red light. Hypersensitivity is lost in a *phyA* null background (i.e. *phyA* is epistatic to *spa1*), indicating a role for SPA1 that is dependent on and specific to the phyA receptor. The sequence of the *spa1* locus is also available.[48] The *SPA1* gene encodes a protein with homology to kinases involved in signal transduction. It also contains a WD-repeat motif, a structure found in many signalling proteins. A similar phenotype to the *spa1* mutant is displayed by the recently published *eid1* mutant.[49] This mutation also causes hypersensitivity to red and far-red light, and *phyA* null mutants are again epistatic to it. The product of this locus may well be involved in the same processes as *SPA1*, although we do not yet have the sequence of the gene involved.

While *spa1*, *eid1*, *far1*, *fhy1*, *fhy3*, *fin2* and *pat1* all show phenotypes specific to phyA signalling, it is this reviewer's view that this pathway has still not been screened to saturation.

Other phytochrome signalling mutants

The isolation of mutations that specifically affect the signalling pathways of other phytochromes presents problems that do not apply to the phyA signalling mutant screens outlined above. First, phyA, phyB, phyD and phyE seem to form a redundant system for red-light perception.[28–30] Consequently, any mutation causing a red-light specific phenotype may be affecting a pathway common to several phytochromes, whereas a far-red phenotype alone indicates specificity to phyA. Secondly, red light has many effects on plant development which are not directly mediated by phytochromes, such as the conversion of protochlorophyllide to chlorophyll and the activation of photosynthesis. As a hypothetical example, a mutant that has a specific effect on the response of the hypocotyl to sugar availability could have a phenotype specific to seedlings grown in red or white light. The elongation of the hypocotyl is known to respond to sugar levels,[50] and the sugars produced by photosynthesis under red light could produce a light-dependent phenotype. For these reasons, it is unwise to interpret a long hypocotyl under red light as a sole indicator of a mutation in a gene encoding a component specific to phyB signalling.

Despite the above objections, it is possible to imagine an excellent candidate for a phyB-signalling mutant. Such a mutant would show an elongated phenotype equivalent to a *phyB* null mutant, *phyB* would be completely epistatic to it, and it would cause the loss of classic red/far-red photoreversible low-fluence responses. Such a mutant would be the direct phyB equivalent of phyA signalling mutant such as *fhy3*. However, such a mutant has never been isolated, despite many extensive screens in different labs and the ease with which the phenotype would be detected. The failure to isolate a complete or near-complete loss-of-function phyB-signalling mutant probably indicates redundancy in this pathway, a recurring theme in light signal perception and transduction in plants.

Putative phyB signalling mutants are known, however (Table 3). The previously characterized red-light specific loss-of-sensitivity mutants are *red1*, *pef2* and *pef3*.[38, 51] They show something of the phenotype expected of a weak *phyB* allele, a long hypocotyl specifically under red light. They are likely to be involved in photomorphogenesis, but their specificity to phyB signalling is unproven; consequently they could be directly involved in transmission of photomorphogenic signals from the photoreceptor, or their action could be some distance downstream. Until we know the molecular functions of

the genes involved, and the phenotypes of their double mutants with each other and with *phyB*, we can tell little about phyB signalling from these mutants. The *early flowering 3 (elf3)* mutant also shows a long hypocotyl phenotype in red light, indicating a possible role in phyB signalling. However, the synergistic phenotype of the *phyB elf3* double mutant implies that these genes may operate in parallel.[52]

Although no proven loss-of-function phyB signalling mutants exist, the *poc1* mutant[53] appears to be a gain-of-function phyB-signalling mutant to which *phyB* is epistatic. The mutation, a T-DNA insertion, is in the promoter of the *PIF3* gene. The PIF3 protein is known to interact specifically with the light-activated conformation of phyB, Pfr[54] which makes *poc1* an excellent candidate for an upstream phyB-signalling mutant.

However, PIF3 also interacts with phyA[55] and the *poc1* mutation in the *PIF3* promoter appears to cause a red-light dependent increase in transcript level.[53] The epistasis of *phyB* to *poc1* may not therefore indicate a specific role of PIF3 in phyB signalling. (See also, the article by Quail, this volume.)

Inferences

The revelation that phytochrome B can interact directly with a transcription factor bound to DNA in a light-dependent manner[56] makes a direct role of phytochromes in the control of gene expression a distinct possibility. If phytochrome interacts directly with one or more transcription factors, we might not expect to see many (if a single factor such as PIF3 is involved) or any (if phytochrome has multiple, redundant, or essential targets) phytochrome signalling mutants. However, phytochromes, due to their size, are not free to diffuse into the cellular nucleus. They are, however, known to be translocated to the nucleus in a light-dependent manner.[57–59] This light-regulated nuclear translocation system is itself likely to be a multi-step signalling process. Consequently, at least some of the phytochrome signalling mutations described above are likely to affect the process of nuclear translocation. The transport of proteins to and from the nucleus is also implicated in other aspects of the control of photomorphogenic development.[60–62] Once located in the nucleus, phytochromes may control the activities of other components by means of their intrinsic kinase activity.[63] Such an enzymatic role for phytochromes implies that they may have multiple, possibly redundant protein substrates; hence, mutations affecting these substrates may not be clearly visible in genetic screens. (See also, the articles by Nagy *et al.* and Fankhauser, this volume.)

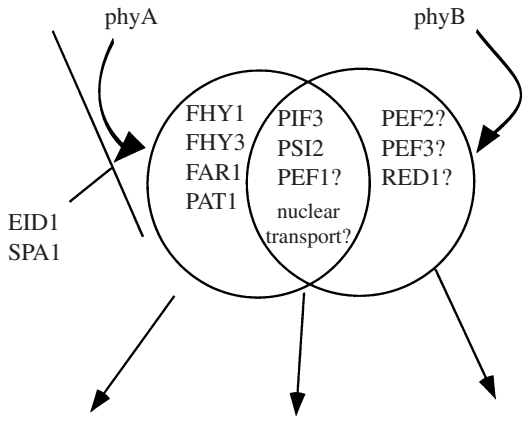

Figure 3. Figure summarising current knowledge of phytochrome signalling pathways. Speculation is kept to a minimum.

Now we know the central role of nuclear transport in phytochrome signalling, it is a priority to characterize the effect of the known mutations on nuclear translocation of phytochromes. It is also important to investigate the sub-cellular localization of potential phyA signalling components. This approach has already led to the discovery that while SPA1 and FAR1 are nuclear localized, PAT1 is cytoplasmic, implying an early role for PAT1 in phyA signalling (Figure 2). The cytoplasmic steps in phyA signalling are likely to be the earliest, as a light-induced translocation of phyA to the nucleus is probably required before nuclear components become involved.[58] It is possible therefore to place some of the components of phyA signalling in a highly speculative order (Figure 2). However, this is certainly an over-interpretation of what we actually know.

The relationship between the selected phytochrome signalling mutants is better represented by a Venn diagram (Figure 3), showing that we know the specificities of some of the components, but have little idea of their order. Ordering this pathway in the way in which mutants in other pathways can be ordered, i.e. epistasis[64, 65] is difficult, as all the phenotypes involved affect the same character (light response) to differing degrees. In addition, none of the signal transduction mutants provides a total block of any given response, as does, for example, the *phyA* photoreceptor mutant in the response of seedlings to far-red light. A study of the epistatic relations between the various signal transduction mutants described here may still yield useful data, however, if the mutants were gathered together in a single lab, and, more laboriously, in a single ecotype background. However, the likely way

in which our knowledge of this pathway will progress is that the signalling mutant loci will be cloned, and the molecular functions and interactions of their products characterized.

Note added in proof

Since this review went to press, three papers of significant relevance have been published. All three of the papers describe new mutants which specifically affect phyA signalling. The *fin219* mutant[66], a new member of the far-red-specific hyposensitive mutant class, has a lesion at a locus encoding an auxin-induced, GH3-like gene. The *rsf1*[67] and *hfr1*[68] mutants, which map very close to each other and may therefore be allelic, are also hyposensitive to far-red light. The *hfr1* locus has been cloned and found to encode a basic helix–loop–helix protein that interacts with PIF3.

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