PLANT GENETICS

Light in the dark

Elliot M. Meyerowitz

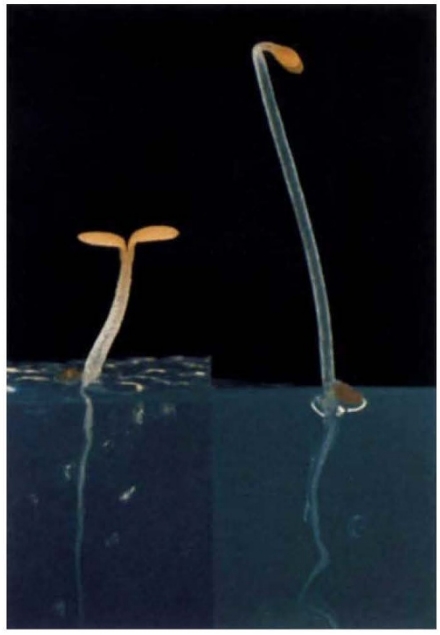
WRITING in *Cell*[1], X.-W. Deng and colleagues describe the cloning of one of a particular class of genes concerned in the control of a plant's response to light. That achievement, and the authors' related observations, will help in the endeavour to understand plant phototransduction. The paper is also the latest in a series reporting the association of familiar functional modules of regulatory proteins in unfamiliar combinations; this is a line of plant research that is turning in a number of surprises and will be of interest to a much wider community.

Rather than using neurons and muscles to respond to the environment, plants have evolved plastic forms of development that use cell division, elongation and differentiation for the purpose. Among the environmental stimuli with the most dramatic effects is light. A plant grown in the dark comes to have elongated stems, small leaves and undifferentiated chloroplasts (it is white). The long stems are thought to be an adaptive response, an attempt by a shaded or underground plant to move its growing point to a sunnier environment.

Several laboratories have been attempting to understand this type of light control of development by looking for mutants in which such control is absent, causing plants to behave in the light as if they were in the dark or vice versa. Both types of mutants have been found. An example of a plant that acts (in one way) as if it were in the dark, regardless of its illumination, is the *HY3* mutant of *Arabidopsis thaliana*[2]. This mutant germinates normally, but begins growth with an overly long stem and does not become fully green. Molecular evidence shows that the *HY3* gene codes for the apoprotein of one of the *Arabidopsis* phytochromes[3,4], the photoreceptors for long-wavelength light.

A more puzzling class of mutants is that in which dark-grown plants behave as if they were in the light[5–7]. The existence of recessive mutants of this type implies that the effect of light is to inactivate repressors of photomorphogenesis. If so, cloning these genes should reveal some of the mechanisms by which phytochromes (and the other plant photoreceptors) act.

The first gene in this class has now been cloned by Deng *et al.*, from *Arabidopsis*[1]. The gene is called *COP1* (for constitutively photomorphogenic); the recessive mutant phenotype is that seedlings grown in the dark (see figure) have a morphology and form of cellular differentiation similar to that of plants grown in the light. Intriguingly, *COP1* codes for a protein with two familiar motifs: it has a zinc-finger region near its amino terminus, and four copies of the WD-40 repeat towards its carboxy-terminal end. The WD-40 repeat is of unknown function, but is found in a variety of regulatory proteins[8,9], including heterotrimeric G protein β-subunits (G_β) and the yeast transcriptional repressor TUP1.



A four-day-old dark-grown *COP1* mutant seedling of *Arabidopsis thaliana* (left) in comparison with a dark-grown wild-type seedling of the same age (right). (Courtesy of X.-W. Deng.)

This finding[1] does not reveal the mechanism of plant phototransduction, but it does raise several interesting possibilities. The WD-40 proteins emphasized by Deng *et al.* are TUP1 and G_β. TUP1 acts as a transcriptional repressor in association with another protein, SSN6 (ref. 10). These proteins are thought to be recruited to their target promoters by additional, sequence-specific DNA-binding proteins. This is certainly consistent with the proposed function of COP1, that it acts as a repressor of the photomorphogenic pathway. The similarity of COP1 to the β-subunit of heterotrimeric G proteins is also consistent with evidence that light responses mediated by phytochrome and blue light may act through G proteins[11,12], as do rhodopsin-mediated light responses in animals.

Quite a different possibility is that the COP1 protein is a regulated component of a specific RNA-splicing pathway. Yeast PRP4, a part of the U4/U6 small nuclear ribonucleoprotein, is a WD-40 protein[13,14]; and PRP11 (RNA11), a protein that is part of the spliceosome, has a zinc-finger region[15]. Both proteins are required for RNA splicing, PRP4 early in spliceosome assembly, and PRP11 as a spliceosome component. So these motifs are known to act together (though on different protein molecules) in processing pre-messenger RNA. Could they function together, on the same protein molecule, to regulate light-specific RNA-splicing patterns in plants?

The finding of hitherto unknown associations of two familiar protein domains is becoming a regular result of the cloning of plant genes. Among the other examples are the *Arabidopsis* homeobox leucine-zipper proteins[16,17], and the calcium-dependent protein kinase of soybean that contains a calmodulin motif[18]. Whether similar proteins to these exist in animals is still unknown. One possibility is that they do. Another is that plants and animals have similar functional protein motifs, inherited as part of their prokaryotic or unicellular eukaryotic legacy, but that they use these motifs in different combinations.

One of the main issues raised by the parallel study of plant and animal development is the extent to which developmental processes in the two kingdoms resemble each other. Each kingdom, as far as we know, evolved multicellular development independently. Is there only one way to do it? Or are the solutions to common developmental problems based on commonly inherited and ancient protein domains, used in very different ways? These are profound questions, the answers to which will eventually come from continued cloning and sequencing of developmentally important genes such as *COP1*. □

Elliot M. Meyerowitz is in the Division of Biology, California Institute of Technology, Pasadena, California 91125, USA.

1. Deng, X.-W. *et al. Cell* **71**, 791–801 (1992).
2. Koorneef, M. *et al. Z. PflanzenPhysiol.* **100**, 147–160 (1980).
3. Nagatani, A. *et al. Pl. Cell Physiol., Tokyo* **32**, 1119–1122 (1991).
4. Somers, D. E. *et al. Plant Cell* **3**, 1263–1274 (1991).
5. Chory, J. *et al. Cell* **58**, 991–999 (1989).
6. Chory, J. & Poto, C. A. *Proc. natn. Acad. Sci. U.S.A.* **87**, 8776–8780 (1990).
7. Deng, X.-W. *et al. Genes Dev.* **5**, 1172–1182 (1991).
8. Fong, H. K. W. *et al. Proc. natn. Acad. Sci. U.S.A.* **83**, 2162–2166 (1986).
9. van der Voorn, L. & Ploegh, H. L. *FEBS Lett.* **307**, 131–134 (1992).
10. Keleher, C. A. *et al. Cell* **68**, 709–719 (1992).
11. Bossen, M. E. *et al. Physiologia Pl.* **80**, 55–62 (1990).
12. Warpeha, K. M. F. *et al. Proc. natn. Acad. Sci. U.S.A.* **88**, 8925–8929 (1991).
13. Bjorn, S. P. *et al. Molec. cell. Biol.* **9**, 3698–3709 (1989).
14. Banroques, J. & Abelson, J. N. *Molec. cell. Biol.* **9**, 3710–3719 (1989).
15. Chang, T.-H. *et al. Molec. cell. Biol.* **8**, 2379–2393 (1988).
16. Ruberti, I. *et al. EMBO J.* **10**, 1787–1791 (1991).
17. Schena, M. & Davis, R. W. *Proc. natn. Acad. Sci. U.S.A.* **89**, 3894–3898 (1992).
18. Harper, J. F. *et al. Science* **252**, 951–954 (1991).