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REVIEW ARTICLE

Ultraviolet Radiation and Plants: Burning Questions

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INTRODUCTION

Plants use sunlight for photosynthesis and, as a consequence, are exposed to the ultraviolet (UV) radiation that is present in sunlight. UV radiation is generally divided into three classes: UV-C, UV-B, and UV-A. The UV-C region of the UV spectrum includes wavelengths below 280 nm; these highly energetic wavelengths are effectively absorbed by ozone in the stratosphere and, thus, are not present in sunlight at the earth's surface. UV-C wavelengths will be removed from the light reaching the earth's surface so long as there is any ozone present (Caldwell et al., 1989). In contrast, UV radiation in the UV-B region, from 280 to 320 nm, does reach ground level. The UV-B portion of sunlight has received much attention in recent years because irradiation from this spectral region (especially 297 to 310 nm) will increase as the stratospheric ozone concentration decreases (Caldwell et al., 1989). Currently, ozone decreases result from chlorofluorocarbon contamination of the stratosphere (McFarland and Kaye, 1992). UV wavelengths from 320 to 390 nm, which make up the UV-A region of the spectrum, are not attenuated by ozone, so their fluence will be unaffected by ozone layer reduction.

Like all living organisms, plants sense and respond to UV radiation, both the wavelengths present in sunlight (UV-A and UV-B) and the wavelengths below 280 nm (UV-C). All types of UV radiation are known to damage various plant processes. Such damage can be classified into two categories: damage to DNA (which can cause heritable mutations) and damage to physiological processes. There has been much speculation about how increased UV radiation exposure will affect plants, but as yet, there are no definitive answers. In this review, I will discuss the kinds of damage that UV radiation can inflict on plants, the mechanisms plants use to perceive and respond to UV radiation, and the ecological relevance of UV light wavelengths that have been used in the experimental analysis of plant responses to UV radiation.

DNA DAMAGE AND REPAIR IN PLANTS

UV-C radiation has been used as a mutagenic agent in plants, and it is known to reactivate the maize *Mutator* transposable element (Walbot, 1992). To prevent mutation and/or cell death, UV radiation–induced DNA damage must be repaired before DNA replication. Repair of UV radiation–induced lesions may be of particular importance in plant pollen, especially in wind-pollinated species (Jackson, 1987). DNA damage must also be repaired to allow transcription (Sauerbier and Hercules, 1978).

Although UV-C damage is not physiologically relevant for plants growing in the sun, short-wavelength (UV-C) radiation from germicidal lamps has often been used to study DNA damage in animals and bacteria, as well as in plants. UV-C has been used because DNA has a strong absorption maximum in the UV-C range (at 260 nm); UV-C photons are highly energetic, and high levels of damage can thus be created quickly. Also, high-output UV-B radiation sources and spectroradiometers are expensive.

The best-studied UV radiation–induced DNA lesion is the cyclobutane-type pyrimidine dimer (CPD). Other types of DNA damage are the pyrimidine(6,4)pyrimidone dimer, diverse rare DNA photoproducts, and indirect types such as DNA–protein crosslinks and singlet oxygen damage (Peak and Peak, 1986). Several investigators have measured CPD DNA damage directly in plants or in plant cell cultures (McLennan, 1987; Pang and Hays, 1991; Quaite et al., 1992), but no other types of UV radiation–induced DNA damage have been reported in plants.

UV radiation–induced DNA damage can be repaired by three mechanisms: photoreactivation, excision repair, or recombinational repair (Smith, 1989; Kornberg and Baker, 1992). CPDs can be repaired by all three methods, but the other UV radiation–induced DNA lesions can be repaired only by excision or recombinational repair.

During photoreactivation repair, CPDs are monomerized by the enzyme photolyase. One distinctive characteristic of photolyases, including those that have been detected in plants (McLennan, 1987; Pang and Hays, 1991), is that they require 370 to 450 nm radiation as an energy source. Thus, CPD-type DNA damage is implicated in any UV radiation response that can be reversed by irradiation with 370 to 450 nm "photoreactivating" radiation. By this criterion, CPD formation is involved in UV-C–induced coumestrol production in *Phaseolus* (Beggs et al., 1985), induction of chromosomal aberrations in barley

root tip cells (Cieminis et al., 1987), induction of DNA repair synthesis in petunia pollen (Jackson, 1987), and the inhibition of anthocyanin production in sorghum internodes (Hashimoto et al., 1991). Photoreactivation can be confirmed by measuring the concentration of UV radiation–induced CPDs before and after 370 to 450 nm light treatment; these measurements have not been reported for any of the above plant studies.

Other types of UV radiation–induced DNA base damage can be repaired by excision. Excision repair can be divided into three steps: nicking of the damaged DNA near the site of the damage, removal of multiple bases in the damaged strand, and resynthesis to fill the gap. Excision has been measured in plant cells (McLennan, 1987), and an endonuclease that nicks DNA that contains CPDs has been partially purified from carrot cells (McLennan and Eastwood, 1986).

The third type of DNA repair, recombinational repair, has not been reported in plants. In this repair pathway, DNA lesions are bypassed during DNA replication, and the resulting gaps are filled in later using information from the sister duplex (Kornberg and Baker, 1992). Because most DNA replication in plants occurs in apical meristems and the surrounding cells, which are usually shielded from the sun by many layers of tissue, recombinational repair may not be particularly important in the repair of UV radiation damage in plants. Other, as yet undiscovered, repair strategies may exist in plants.

PLANT PHYSIOLOGICAL RESPONSES TO UV-B RADIATION

Many different plant responses to supplemental UV-B radiation have been observed (Tevini and Teramura, 1989). The best studies have increased UV-B levels to simulate conditions that would exist with a defined reduction in the ozone layer (typically 10 to 20%). As a control, the same lamps are shielded with a plastic film that absorbs all UV-B wavelengths. Thus, all parts of the spectrum except the UV-B region are held constant. Although with this system it is possible to determine whether a response results from supplemental UV-B, the mechanism by which that response occurs may not be obvious. For instance, changes seen after supplemental UV-B radiation include biomass reductions (Tevini et al., 1981; Lydon et al., 1986; Sullivan and Teramura, 1988), decreases in the percentage of pollen germination (Flint and Caldwell, 1984), changes in the ability of crop plants to compete with weeds (Barnes et al., 1990), epidermal deformation (Tevini et al., 1981), changes in cuticular wax composition (Tevini and Steinmuller, 1987), and increased flavonoid levels (Tevini et al., 1981, 1991a; Beggs and Wellman, 1985).

These changes could result from any number of primary UV-B events: DNA damage, direct photosynthetic damage, membrane changes, protein destruction, hormone inactivation (Tevini et al., 1989, 1991b), signal transduction through phytochrome (which photoconverts in response to UV-B) (Pratt and Butler, 1970), or signal transduction via a UV-B photoreceptor. To determine precisely which factor or factors are involved, the action spectrum (i.e., the level of response at each wavelength) and the kinetics of the response must be established. Figure 1 outlines some steps in determining the mechanism of action of a particular UV radiation response. Fluence-response curves are necessary to create an analytical action spectrum that can identify the chromophore involved (Coohill, 1992). Even if no one factor can be identified as the cause of a UV-B–induced effect (for example, if the action spectrum for plant growth has multiple overlapping peaks), a complex action spectrum can be used to estimate the change expected from a given amount of ozone depletion. Measurement of the lag time between irradiation and appearance of a response can provide information about the mechanism of the response. For example, direct DNA damage is detectable shortly after irradiation, whereas chalcone synthase (CHS) gene expression requires many minutes.

Some detailed mechanistic information is available for five UV-B responses: photomorphogenesis, auxin inactivation, ATPase destruction, photosynthetic damage, and flavonoid induction. Photomorphogenesis is a radiation-induced change in plant form. UV-B enhancement alters the growth of several plant species but does not reduce shoot dry weight (Barnes et al., 1990). An action spectrum of the first positive phototropism (curvature) of the alfalfa hypocotyl has demonstrated that UV-B contributes to the response; plants were kept in red light to isolate this response from the similar response through phytochrome (Baskin and Iino, 1987). A cucumber mutant that lacks light-stable phytochrome (López-Juez et al., 1992) has

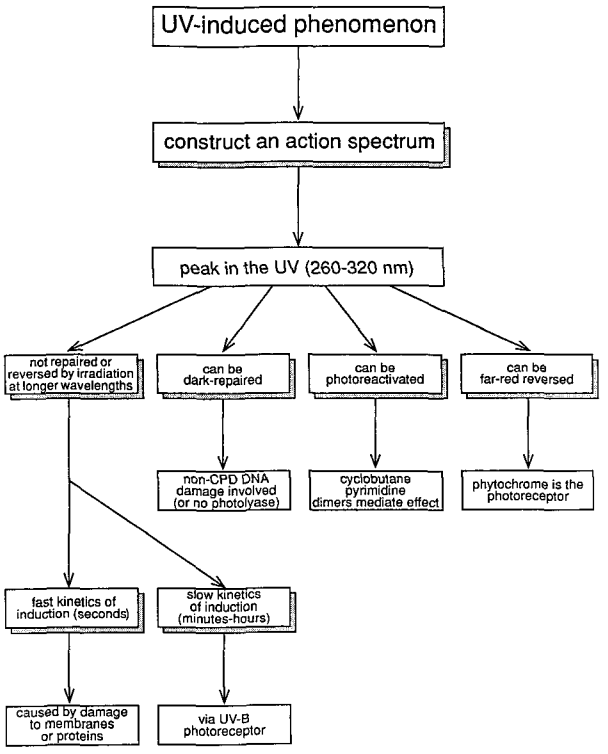

Figure 1. A Decision Tree for UV-Induced Responses.

also been used to measure photomorphogenesis after UV-B treatment; UV-B inhibits hypocotyl growth (Ballare et al., 1991). However, because this mutant has some residual phytochrome function (Whitelam and Smith, 1991), the action of phytochrome in this UV-B response cannot be excluded. In the experiments with cucumber, shielding the actively growing tissues from UV radiation did not affect the magnitude of the decrease in hypocotyl length, so direct effects on cell division or elongation would not explain the UV-B–induced growth inhibition. Recovery after return to uninducing conditions was rapid, again suggesting a true photomorphogenic response to UV-B.

Another UV radiation response is the inhibition of epidermal cell elongation in sunflower seedlings by UV-B and UV-C radiation (Tevini et al., 1989). This inhibition results from the photooxidation of indoleacetic acid to 3-methylenoxidol, which inhibits hypocotyl elongation. The inhibitory effects of photooxidation of indoleacetic acid were seen in vitro and in vivo (Tevini et al., 1989).

A third UV radiation response for which there is some mechanistic information is the destruction of a rose cell plasma membrane ATPase by UV-B radiation. The action spectrum of this response peaks at 290 nm (Murphy, 1983), and the fluence-response curve shows that this response occurs at fluences that are typical of sunlight (Imbrie and Murphy, 1984). The inactivation of ATPase probably results from singlet oxygen–mediated destruction of tryptophan residues in the ATPase protein (Imbrie and Murphy, 1984).

Another well-studied response to UV-B radiation is damage to the photosynthetic apparatus. Photosystem II is the UV-B–sensitive component, but the action spectrum of the UV-B effect does not suggest a specific target molecule (Renger et al., 1989). Damage is assessed by measuring the increase in variable chlorophyll fluorescence; an increase in fluorescence can be observed after doses of UV-B radiation in the physiologically relevant range (Tevini et al., 1991a).

Perhaps the most general plant response to UV-B radiation is activation of the flavonoid biosynthetic pathway. Flavonoids and/or anthocyanins are induced by UV-B exposure in all 14 plant species tested (Tevini et al., 1981; Beggs and Wellman, 1985). Flavonoids and anthocyanins absorb UV radiation, and they generally accumulate in the epidermis, where they could keep UV radiation from reaching photosynthetic tissues (Hahlbrock and Scheel, 1989). The epidermis blocks transmittance of 95 to 99% of incoming UV radiation (Robberecht and Caldwell, 1978). Induction of flavonoids in rye seedlings can prevent UV-B–induced damage to photosynthesis (Tevini et al., 1991a), which suggests that UV radiation protection is one of the functions of these pigments. This could be tested directly using mutants that are defective in the accumulation of flavonoids.

How does UV radiation activate flavonoid expression? Possible receptors for anthocyanin and flavonoid induction after UV-B exposure have been analyzed in sorghum and parsley. The action spectrum for anthocyanin induction in sorghum has several maxima, indicating that both phytochrome and a separate UV-B photoreceptor are involved in anthocyanin induction. There is no peak in the blue region of the spectrum, ruling out the involvement of a blue light photoreceptor. The action spectrum peak (263 nm) for anthocyanin inhibition is photoreactivatible, and this inhibition does not involve phytochrome (Hasimoto et al., 1991). Phytochrome, a blue light photoreceptor, and a UV-B photoreceptor have all been shown to be involved in flavonoid induction in parsley (Bruns et al., 1986); in fact, a white light source that contains all these wavelengths is usually used to induce parsley flavonoids. Feeding riboflavin to parsley cells increases the UV-B induction of CHS, a key enzyme in the flavonoid biosynthesis pathway. This result suggests that the UV-B photoreceptor might be a flavin (Ensminger and Schafer, 1992).

Deletion analysis of the CHS promoter has identified a DNA segment that is responsible for light induction of this gene (Schulze-Lefert et al., 1989; Block et al., 1990). A protein that binds to this DNA segment has also been characterized (Weisshaar et al., 1991). DNA elements in the CHS promoter that allow UV radiation inducibility have also been characterized in petunia and Antirrhinum (Staiger et al., 1989; van der Meer et al., 1990). These DNA elements have in common only a motif that is present in the promoters of many plant genes, not just in light-inducible genes. We still know little about the signal transduction pathway between interception of UV radiation photons and induction of flavonoid biosynthetic gene expression.

Responses to UV-B are also induced by other biotic and abiotic stresses. For example, genes that encode enzymes involved in early steps of flavonoid and phytoalexin biosynthesis, including phenylalanine ammonia-lyase and CHS, are induced by both fungal elicitors and wounding, as well as by UV radiation (Fritzemeier et al., 1983; Hahlbrock and Scheel, 1989). In the case of CHS induction in parsley, fungal elicitor treatment is epistatic to the UV radiation induction (Lozoya et al., 1991), and heat shock overrides both of these (Walter, 1989). CHS may be an integrative branchpoint in phenylpropanoid metabolism.

USE OF UV-C AS A MODEL DAMAGING AGENT

Is UV-C radiation an acceptable model for more ecologically relevant wavelengths in studies of DNA repair or other plant responses? Action spectra for DNA damage in *E. coli* are biphasic in the UV-B range, with a short-wavelength, UV-C–type contribution (from direct DNA damage) and a long-wavelength contribution (from indirect DNA damage) (Peak and Peak, 1986). Provided that UV-B induces both direct and indirect damage in plants as well, and given that UV-C induces direct damage only, UV-C is not a good substitute for UV-B. For example, in animal cells, the contribution of CPDs to total DNA damage falls from 90% at 254 nm to about 50% at 313 nm and to nearly zero at 350 nm (Kantor, 1985).

There is as yet no information about the relative proportions of direct and indirect damage at various wavelengths in plants. However, for higher plants living in sunlight, photoreactivating wavelengths are present in sunlight at such high fluence

that few CPDs should accumulate, provided that the plants contain photolyase. Even if there is a severe depletion of the ozone layer, resulting in increases in the level of shorter wavelength radiation reaching the earth's surface, photoreactivation capacity is sufficient to eliminate CPDs before permanent DNA damage occurs (Beggs et al., 1985). This suggests that direct, non-CPD DNA damage and indirect DNA damage are more ecologically relevant, but these types of damage are not the major products of UV-C radiation. Detection of repair processes in plants should not be affected by these differences, but measurements of repair capacity will be biased when UV-C is used to induce damage. If these limitations are acknowledged, UV-C can be used as a model radiation for studies of certain types of DNA damage and repair in plants.

Is UV-C a useful model for plant responses to UV radiation other than DNA damage and repair? Some UV radiation responses have been measured in both the UV-C and UV-B regions of the spectrum, including tissue damage (Bornman et al., 1986), induction of carotenoids and polyamines (Tevini and Teramura, 1989; Kramer et al., 1991), damage to the photosynthetic apparatus (Kulandaivelu and Noorudeen, 1983), phototropism (Baskin and Iino, 1987), ATPase destruction (Murphy, 1983; Imbrie and Murphy, 1984), unscheduled DNA synthesis in pollen (Jackson, 1987), and anthocyanin and flavonoid induction in parsley, sorghum, and peanut callus (Wellman, 1971; Fritzemeier et al., 1983; Hashimoto et al., 1991). In each case, the response to UV-C differs from the UV-B effect. In the case of tissue and photosynthetic damage, qualitatively different responses to the two types of UV radiation were observed. In the studies of phototropism and ATPase destruction, two separate activities were found, one responsive to UV-C and one to UV-B. Furthermore, UV-C and UV-B have opposite effects on carotenoid levels (Tevini et al., 1981; Campos et al., 1991) and anthocyanin levels (Hashimoto et al., 1991). Therefore, as a general rule, UV-C is not a useful model for physiological responses induced by UV-B.

FUTURE DIRECTIONS

Although many plant responses to UV radiation have been reported, complete mechanistic details of most of these responses have not been elucidated. Certain types of analysis are necessary to fully understand any UV radiation response; a decision tree for analyzing what mechanisms mediate an induction is presented in Figure 1. An action spectrum is critical in defining which chromophore (DNA, a UV-B photoreceptor, cryptochrome, or phytochrome) may be involved and whether more than one chromophore mediates the response. A kinetic analysis is also crucial. The length of the lag time before the appearance of the response can indicate whether the response results directly from photon interaction with a target (e.g., CPD formation) or from changes in gene expression. Other important parameters include determining whether the response can be photoreactivated (if it can, it probably involves CPDs), whether excision repair of the DNA (in the dark) can reverse the effect (in which case it would involve non-CPD DNA damage, provided there is photolyase present), and whether the effect is far-red reversible, a feature diagnostic of phytochrome involvement.

An additional, important approach to dissecting plant responses to UV radiation is genetic. Mutants that are defective in one or more responses to UV radiation and second-site suppressors of these mutants will be very useful in defining the steps in the signal transduction pathway. One step in this direction is a Chlamydomonas mutant that has no photoreactivation capacity (McLennan, 1987). Arabidopsis mutants with increased or decreased sensitivity to UV-B radiation have been found by several laboratories; it would be helpful to have a UV-B photoreceptor mutant analogous to the blue light photoreceptor mutant (Khurana and Poff, 1989).

Increased UV radiation levels from ozone layer reduction will reach plants, and plants will respond. Much more work will be required to define the exact mechanisms of the various UV-B–induced responses. Even with mutants, sound physiology will be required to fully understand each response. Thus, the action spectra and kinetics of any UV-B response should be analyzed. In particular, action spectra of the various kinds of DNA damage and more information about photosensitizers and other ways to produce indirect DNA damage are needed. In addition, many compounds that act as photosensitizers come from plants, but very little is known about the effects of such endogenous photodamaging molecules (Spikes, 1989). It would also be useful to know more about DNA repair mechanisms–rates, capacity, and what enzymes repair what kinds of damage. Finally, two unexplored areas concern tissue and developmental differences in UV radiation responses and how tissue responses are integrated to increase plant survival.

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REFERENCES

Ballare, C.I., Barnes, P.W., and Kendrick, R.E. (1991). Photomorphogenic effects of UV-B radiation on hypocotyl elongation in wild type and stable-phytochrome-deficient mutant seedlings of cucumber. Physiol. Plant. **83,** 652–658.

Barnes, P.W., Flint, S.D., and Caldwell, M.M. (1990). Morphological responses of crop and weed species of different growth forms to ultraviolet-B radiation. Am. J. Bot. **77,** 1354–1360.

Baskin, T.I., and Iino, M. (1987). An action spectrum in the blue and ultraviolet for phototropism in alfalfa. Photochem. Photobiol. **46,** 127–136.

Beggs, C., and Wellman, E. (1985). Analysis of light-controlled anthocyanin formation in coleoptiles of *Zea mays* L.: The role of UV-B, blue, red and far-red light. Photochem. Photobiol. **41,** 481–486.

Beggs, C.J., Stolzer-Jehle, A., and Wellman, E. (1985). Isoflavonoid formation as an indicator of UV stress in bean (*Phaseolus vulgaris* L.) leaves. Plant Physiol. **79,** 630–634.

Block, A., Dangl, J., Hahlbrock, K., and Schulze-Lefert, P. (1990). Functional borders, genetic fine structure, and distance requirements of *cis* elements mediating light responsiveness of the parsley chalcone synthase promoter. Proc. Natl. Acad. Sci. USA **87,** 5387–5391.

Bornman, J.F., Evert, R.F., Mierzwa, R.J., and Bornman, C.H. (1986). Fine structural effects of UV radiation on leaf tissue of *Beta vulgaris*. In Stratospheric Ozone Reduction, Solar Ultraviolet Radiation and Plant Life, R. C. Worrest and M. M. Caldwell, eds (Berlin, Heidelberg: Springer-Verlag), pp. 199–209.

Bruns, B., Hahlbrock, K., and Schafer, E. (1986). Fluence dependence of the ultraviolet-light–induced accumulation of chalcone synthase mRNA and effects of blue and far-red light in cultured parsley cells. Planta **169,** 393–398.

Caldwell, M.M., Teramura, A.H., and Tevini, M. (1989). The changing solar ultraviolet climate and the ecological consequences for higher plants. Trends Ecol. Evol. **4,** 363–367.

Campos, J.L., Figueras, X., Pinol, M.T., Boronat, A., and Tiburcio, A.F. (1991). Carotenoid and conjugated polyamine levels as indicators of ultraviolet-C induced stress in *Arabidopsis thaliana*. Photochem. Photobiol. **53,** 689–693.

Cleminis, K.G.K., Ranceliene, V.M., Prijalgauskiene, A.J., Tiunaitiene, N.V., Rudzianskaite, A.M., and Jancys, Z.J. (1987). Chromosome and DNA damage and their repair in higher plants irradiated with short-wave ultraviolet light. Mutat. Res. **181,** 9–16.

Coohill, T.P. (1992). Action spectra revisited. J. Photochem. Photobiol. B. **13,** 95–100.

Ensminger, P. A., and Schafer, E. (1992). Blue and ultraviolet-B light photoreceptors in parsley cells. Photochem. Photobiol. **55,** 437–447.

Flint, S.D., and Caldwell, M.M. (1984). Partial inhibition of in vitro pollen germination by simulated solar ultraviolet-B radiation. Ecology **65,** 792–795.

Fritzemeier, K.-H., Rolfs, C.-H., Pfau, J., and Kindl, H. (1983). Action of ultraviolet-C on stilbene formation in callus of *Arachis hypogaea*. Planta **159,** 25–29.

Hahlbrock, K., and Scheel, D. (1989). Physiology and molecular biology of phenylpropanoid metabolism. Annu. Rev. Plant Physiol. Plant Mol. Biol. **40,** 347–369.

Hashimoto, T., Shichijo, C., and Yatsuhashi, H. (1991). Ultraviolet action spectra for the induction and inhibition of anthocyanin synthesis in broom sorghum seedlings. J. Photochem. Photobiol. B. **11,** 353–363.

Imbrie, C.W., and Murphy, T.M. (1984). Mechanism of photoinactivation of plant plasma membrane ATPase. Photochem. Photobiol. **40,** 243–248.

Jackson, J.F. (1987). DNA repair in pollen. A review. Mutat. Res. **181,** 17–29.

Kantor, G.J. (1985). Effects of sunlight on mammalian cells. Photochem. Photobiol. **41,** 741–746.

Khurana, J.P., and Poff, K.L. (1989). Mutants of *Arabidopsis thaliana* with altered phototropism. Planta **178,** 400–406.

Kornberg, A., and Baker, T.A. (1992). DNA Replication (New York: W.H. Freeman), pp. 771–791.

Kramer, G.F., Norman, H.L., Krizek, D.T., and Mirecki, R.M. (1991). Influence of UV-B radiation on polyamines, lipid peroxidation and membrane lipids in cucumber. Phytochemistry **30,** 2101–2108.

Kulandaivelu, G., and Noorudeen, A.M. (1983). Comparative study of the action of ultraviolet-C and ultraviolet-B on photosynthetic electron transport. Physiol. Plant. **58,** 389–394.

López-Juez, E., Nagatani, A., Tomizawa, K.-I., Deak, M., Kern, R., Kendrick, R.E., and Furuya, M. (1992). The cucumber long hypocotyl mutant lacks a light-stable PHYB-like phytochrome. Plant Cell **4,** 241–251.

Lozoya, E., Block, A., Lois, R., Hahlbrock, K., and Scheel, D. (1991). Transcriptional repression of light-induced flavonoid synthesis by elicitor treatment of cultured parsley cells. Plant J. **1,** 227–234.

Lydon, J., Teramura, A.H., and Summers, E.G. (1986). Effects of ultraviolet-B radiation on the growth and productivity of field grown soybean. In Stratospheric Ozone Reduction, Solar Ultraviolet Radiation and Plant Life, R.C. Worrest and M.M. Caldwell, eds (Berlin, Heidelberg: Springer-Verlag), pp. 313–325.

McFarland, M., and Kaye, J. (1992). Chlorofluorocarbons and ozone. Photochem. Photobiol. **55,** 911–929.

McLennan, A.G. (1987). DNA damage, repair, and mutagenesis. In DNA Replication in Plants, J.A. Bryant and V.L. Dunham, eds (Boca Raton, FL: CRC Press), pp. 135–186.

McLennan, A.G., and Eastwood, A.C. (1986). An endonuclease activity from suspension cultures of *Daucus carota* which acts upon pyrimidine dimers. Plant Sci. **46,** 151–157.

Murphy, T.M. (1983). Membranes as targets of ultraviolet radiation. Physiol. Plant. **58,** 381–388.

Pang, Q., and Hays, J.B. (1991). UV-B–inducible and temperature-sensitive photoreactivation of cyclobutane pyrimidine dimers in *Arabidopsis thaliana*. Plant Physiol. **95,** 536–543.

Peak, M.J., and Peak, J.G. (1986). Molecular photobiology of UVA. In The Biological Effects of UVA Irradiation, F. Urbach and R.W. Gange, eds (Westport, CT: Praeger), pp. 42–52.

Pratt, L.H., and Butler, W.L. (1970). Phytochrome conversion by ultraviolet light. Photochem. Photobiol. **11,** 503–509.

Quaite, F.E., Sutherland, B.M., and Sutherland, J.C. (1992). Action spectrum for DNA damage in alfalfa lowers predicted impact of ozone depletion. Nature **358,** 576–578.

Renger, G., Volker, M., Eckert, H.J., Fromme, R., Hohm-Veit, S., and Graber, P. (1989). On the mechanism of photosystem II deterioration by UV-B irradiation. Photochem. Photobiol. **49,** 97–105.

Robberecht, R., and Caldwell, M.M. (1978). Leaf epidermal transmittance of ultraviolet radiation and its implication for plant sensitivity to ultraviolet-radiation induced injury. Oecologia **32,** 277–287.

Sauerbier, W., and Hercules, K. (1978). Gene and transcription unit mapping by radiation effects. Annu. Rev. Genet. **12,** 329–363.

Schulze-Lefert, P., Becker-André, M., Schulz, W., Hahlbrock, K., and Dangl, J.L. (1989). Functional architecture of the light-responsive chalcone synthase promoter from parsley. Plant Cell **1,** 707–714.

Smith, K.C. (1989). UV radiation effects: DNA repair and mutagenesis. In The Science of Photobiology, K.C. Smith, ed (New York: Plenum Press), pp. 111–134.

Spikes, J.D. (1989). Photosensitization. In The Science of Photobiology, K.C. Smith, ed (New York: Plenum Press), pp. 79–110.

Staiger, D., Kaulen, H., and Schell, J. (1989). A CACGTG motif of the *Antirrhinum majus* chalcone synthase promoter is recognized by an evolutionarily conserved nuclear protein. Proc. Natl. Acad. Sci. USA **89,** 6930–6934.

Sullivan, J.H., and Teramura, A.H. (1988). Effects of ultraviolet-B irradiation on seedling growth in the *Pinaceae*. Am. J. Bot. **75,** 225–230.

Tevini, M., and Steinmuller, D. (1987). Influence of light, UV-B radiation, and herbicides on wax biosynthesis of cucumber seedlings. J. Plant Physiol. **131,** 111–121.

Tevini, M., and Teramura, A.H. (1989). UV-B effects on terrestrial plants. Photochem. Photobiol. **50,** 479–487.

Tevini, M., Iwanzik, W., and Thoma, U. (1981). Some effects of enhanced UV-B irradiation on the growth and composition of plants. Planta **153,** 388–394.

Tevini, M., Braun, J., Grusemann, P., and Ros, J. (1989). UV-Wirkungen auf Nutzpflantzen. In Akad. Natursch. Landschaftspfl, (Laufen/Salzach, Germany), pp. 38–52.

Tevini, M., Braun, J., and Fieser, G. (1991a). The protective function of the epidermal layer of rye seedlings against ultraviolet-B radiation. Photochem. Photobiol. **53,** 329–333.

Tevini, M., Mark, U., Fieser, G., and Saile, M. (1991b). Effects of enhanced solar UV-B radiation on growth and function of selected crop plant seedlings. In Photobiology, E. Riklis, ed (New York: Plenum Press), pp. 635–649.

van der Meer, I.M., Spelt, C., Mol, J.N.M., and Stuitje, A.R. (1990). Promoter analysis of the chalcone synthase (*chsA*) gene of *Petunia hybridia*: A 67 bp promoter region directs flower-specific expression. Plant Mol. Biol. **15,** 95–109.

Walbot, V. (1992). Reactivation of *Mutator* transposable elements of maize by ultraviolet light. Mol. Gen. Genet., in press.

Walter, M.H. (1989). The induction of phenylpropanoid biosynthetic enzymes by ultraviolet light or fungal elicitor in cultured parsley cells is overridden by a heat-shock treatment. Planta **177,** 1–8.

Weisshaar, B., Armstrong, G.A., Block, A., da Costa e Silva, O., and Hahlbrock, K. (1991). Light-inducible and constitutively expressed DNA binding proteins recognizing a plant promoter element with functional relevance in light responsiveness. EMBO J. **10,** 1777–1786.

Wellman, E. (1971). Phytochrome-mediated flavone glycoside synthesis in cell suspension cultures of *Petroselinum hortense* after preirradiation with ultraviolet light. Planta **101,** 283–286.

Whitelam, G.C., and Smith, H. (1991). Retention of phytochrome-mediated shade avoidance responses in phytochrome-deficient mutants of *Arabidopsis*, cucumber and tomato. J. Plant Physiol. **139,** 119–125.