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Update on Plant Stress

Repair of DNA Damage Induced by Ultraviolet Radiation

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Studies documenting the depletion of the ozone layer and the resulting increases in UV-B radiation (280–320 nm) at the Earth's surface have served to focus attention on the biological effects of UV light. One obvious target for UV-B-induced damage is DNA. Although all biological tissues are rich in UV-absorbing agents (largely nucleic acids and proteins) and plants produce additional UV-absorbing pigments, no DNA in superficial tissue can completely avoid UV exposure. Plants, like all living organisms, must have some capacity for the repair of UV-induced DNA damage. Because plants are unique in the obligatory nature of their exposure to UV, it is also conceivable that they may have evolved particularly efficient mechanisms for the elimination of UV-induced DNA damage. This review will summarize what we know about DNA repair mechanisms in higher plants. Readers interested in broader aspects of UV-induced damage and UV filters are directed to recent reviews (Middleton and Teramura, 1994; Strid et al., 1994; Fiscus and Booker, 1995). Our knowledge of DNA repair mechanisms in plants lags far behind our understanding of these pathways in animals, and a significant number of questions concerning the basic phenomenology of DNA repair in plants remain to be addressed.

UV-INDUCED DNA DAMAGE HAS BOTH TOXIC AND MUTAGENIC EFFECTS

The CPD and the pyrimidine (6–4) pyrimidinone dimer (the 6–4 photoproduct) make up approximately 75 and 25%, respectively, of the UV-induced DNA damage products (Fig. 1) (Mitchell and Nairn, 1989). The biological effects of these lesions have been extensively studied in microbial and mammalian systems, where UV-induced DNA damage has been shown to produce two distinct effects: mutagenesis and toxicity. At the molecular level, pyrimidine dimers have been shown to inhibit the progress of microbial and mammalian DNA polymerases. When the advancing polymerase reaches one of these lesions during replication, the enzyme will attempt to install a nucleotide opposite the lesion, only to recognize this as a mismatched base. The newly installed base is then excised by the 3′ to 5′ mismatch repair exonuclease function of the polymerase. Because pyrimidine dimers cannot effectively base pair with other nucleotides, they are not directly mutagenic, but instead act as blocks to DNA replication. It is interesting, and very significant in terms of UV-induced toxicity, that RNA polymerase has also been shown to "stall" at both CPDs and 6–4 photoproducts (Protic-Sabljic and Kraemer, 1986; Mitchell et al., 1989). In the absence of repair a single pyrimidine dimer is sufficient to completely eliminate expression of a transcriptional unit. In addition, evidence suggests that the stalled RNA polymerase II remains bound to the site of the obstruction (Donahue et al., 1994). Because of this, it is possible that persisting lesions may actually reduce the overall concentration of free RNA polymerase. Because every pyrimidine dimer can act as a block to transcription and replication, whereas only a small fraction of dimers result in a mutation (see below), the inhibitory effects of UV on transcription and replication are probably more significant (in terms of cell growth) than its mutagenic effects. Any living tissue, even one in which cell division does not occur, has to be able to either avoid or repair UV-induced DNA damage if it is to survive.

It is important to note, however, that the ability of UV-induced pyrimidine dimers to act as blocks to both RNA and DNA polymerase has never been demonstrated in a plant system. Because the developmental pattern of plants makes them unusually resistant to the consequences of mutagenesis (Walbot, 1985), it is conceivable that plants might, to some extent, sacrifice accuracy in DNA replication to reduce the toxicity of UV-induced damage. In addition, because of the small size of their introns, plant transcriptional units tend to be quite small, averaging perhaps one-fourth the size of those of animals. As a result, the probability of a dimer occurring within a transcriptional unit is reduced proportionally, thus enhancing the intrinsic resistance of plant cells to the inhibitory effects of UV on transcription.

PATHWAYS FOR TOLERANCE OF UV-INDUCED DNA DAMAGE: MUTAGENESIS AND RECOMBINATION

UV-induced pyrimidine dimers are known to block replication of DNA. DNA replication will normally reinitiate 3′ to the lesion, but a gap remains in the newly synthesized daughter strand at the site opposite the DNA damage product. Alternate pathways for the synthesis of DNA that enable cells to fill in these daughter-strand gaps in spite of the continued presence of dimers have been observed in some microbes and animals. Mechanisms that reduce the toxic effects of DNA damage products without actually repairing the damage are referred to as "tolerance" mech-

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Abbreviation: CPD, cyclobutyl pyrimidine dimer.

Figure 1. Observed pathways for the repair and tolerance of UV-induced DNA damage products in higher plants. The toxicity and/or mutagenicity of any particular type of lesion is determined by its rate of induction, the immediate biological effects of its persistence, and both the efficiency and mechanism of tolerance and repair pathways. "Yes" indicates that this mode of repair has been documented in at least one higher plant species. "?" indicates that this pathway, although observed in microbial and/or animal systems, has not yet been demonstrated in plants.

	cyclobutyl dimer	pyrimidine (6-4) pyrimidinone dimer	Minor UV-induced photoproducts
Photoreactivation	yes	yes	?
Excision repair	yes	yes	?
Translesion synthesis	?	?	?
Recombinational "repair"	?	?	?

anisms. In *Escherichia coli*, the *umuCD* gene products bind to DNA polymerase and enable it to perform translesion synthesis of DNA (often referred to as "dimer bypass") (Rajagopalan et al., 1992) (Fig. 2A). Because the altered polymerase generally installs adenine residues across from noninformational DNA damage products, cytosine-containing pyrimidine dimers are hotspots for UV-induced C-to-T transitions. Because the *umuCD* gene products are required for translesion synthesis, strains with defects in these genes display an enhanced sensitivity to the lethal effects of UV and a variety of chemical mutagens, yet they completely lack a mutagenic response to these DNA-damaging agents. Translesion synthesis permits DNA replication (and, consequently, enhanced survival) at the expense of accuracy. Similarly, sunlight-induced mutations in humans occur at dipyrimidines, and are primarily C-to-T or CC-to-TT transversions (Ziegler et al., 1994).

It remains to be seen whether UV-induced mutagenesis in plants occurs as a result of dimer bypass. The spectrum of mutations induced through the combined effects of DNA damage, DNA repair, and damage-tolerance mechanisms is only broadly defined in plants, because few UV-induced mutations have actually been sequenced. Because the plant's germline is shielded from UV during the multicellular (seed and adult-plant) stages of growth, studies of UV-induced mutations in higher plants have been limited to the mutagenic effects of UV irradiation of pollen. Mutagenesis of pollen has the advantage of enabling the investigator to observe the induction of mutations such as large deletions, which might otherwise be nontransmissable due to counterselection during the postmeiotic mitoses (although additional selection may occur during the growth of the pollen tube). In fact, UV-induced mutations in maize pollen were generally found to be nontransmissable or to have reduced transmission beyond the first generation, indicating that UV-induced lesions result in large deletions rather than point mutations (Nuffer, 1957). Although this finding suggests that translesion synthesis (which induces point mutations) rarely occurs during repair in pollen or during the early stages of embryonic development, and that UV-induced DNA damage results in chromosome breaks and/or recombination, one must bear in mind that large chromosomal deletions, which result in the simultaneous loss of many genes, are simply easier to score as mutations than single base changes, the majority of which fail to affect gene function. It is also possible that dimer bypass is preferentially employed in somatic cell lines (where mutagenesis is relatively inconsequential), but is not expressed during the critical last stage of pollen development, when mutations can no

Damage tolerance pathways can induce mutagenesis

replication

A: Dimer Bypass

modification of DNA polymerase

B: Recombinational "repair"

(sister chromatid)

Figure 2. DNA damage tolerance pathways. Neither dimer bypass (A) nor recombinational "repair" (B) actually results in the loss of the UV-induced lesion. However, they do permit the cell to complete another round of DNA replication.

longer be eliminated through diplontic selection. Because of its potential role in the creation of genetic diversity (as well as in UV tolerance), more research on translesion synthesis is needed in both plants and animals.

Recombinational "repair" is another tolerance mechanism that permits DNA replication in spite of the persistence of DNA damage products. In contrast to dimer bypass, which involves the synthesis of DNA past a lesion, this pathway instead transfers a preexisting complementary strand from a homologous region of DNA to the site opposite the damage (Fig. 2B). As in the dimer bypass mechanism, the lesion is left unrepaired, but the cell manages to get through another round of replication. When the complementary strand is obtained from the newly replicated sister chromatid, the resulting "repair" is error free. However, if the information is obtained from the homologous chromosome, or perhaps from a similar DNA sequence elsewhere in the genome, there is a possibility that a change will be generated in the gene's sequence either via gene conversion or through the formation of deletions, duplications, and translocations. Although UV irradiation has been shown to induce chromosomal rearrangements in plants (Nuffer, 1957), including homologous intrachromosomal recombination events (Puchta et al., 1995), it remains to be seen whether the filling of daughter-strand gaps via homologous recombination is a significant UV-tolerance mechanism in plants. UV light has been shown to induce previously quiescent transposable elements (Walbot, 1992); it is possible that this effect is the result of chromosomal rearrangements or other repair-related activities.

DIRECT REVERSAL OF UV-INDUCED DNA DAMAGE

In many organisms the biological effects of UV radiation can be significantly reduced by subsequent exposure to light in the blue or UV-A range of the spectrum, a phenomenon known as photoreactivation. In microbes the photoreactivating effects of visible light are due to the actions of the enzyme photolyase. This protein binds specifically to CPDs and, upon absorption of a photon of the appropriate wavelength (350–450 nm), directly reverses the damage in an error-free manner. Photolyases carry two prosthetic groups. One chromophore (either methenyl tetrahydrofolate or 8-hydroxy-5-deazaflavin) absorbs the photoreactivating light and transfers this excitation energy to the other chromophore, a fully reduced FAD^-. FAD^- then transfers an electron to the dimer (Sancar, 1994), inducing its reversal. Evidence for the biological effects of photoreactivation in plants is complicated by the obvious detrimental effects of growing plants in the dark, but this problem can be alleviated to some extent by the use of appropriate controls and of filters that absorb the shorter wavelengths required for photoreactivation (450 nm and under) while transmitting photons of longer photosynthetically active wavelengths. Photoreactivation results in the reversal of several UV-induced phenomena in plants, including mutagenesis, chromosome rearrangements (Ikenaga and Mabuchi, 1966), inhibition of growth, and induction of flavonoid pigments (Beggs et al., 1985). Photolyase activities have been observed in extracts from *Chlamydomonas* and several higher plants, and the action spectrum for reversal of CPDs by the maize and Arabidopsis photolyases has been shown to be similar to that of *E. coli*, a methenyl tetrahydrofolate-type photolyase (Pang and Hays, 1991).

A putative plant photolyase cDNA was recently cloned from wild mustard (Batschauer, 1993), and it displays significant stretches of similarity to previously cloned microbial photolyases. The cDNA hybridizes to an mRNA that is strongly regulated by light; seedlings grown in the dark express low levels of the mRNA, whereas light-grown seedlings express the mRNA at high levels. This result is consistent with earlier observations in beans, where photolyase activities were induced upon exposure of the plants to various light regimens. The bean photolyase was induced 2-fold by a brief exposure to red light; this effect was partially reversed by subsequent exposure to far-red light, suggesting that its induction is phytochrome mediated (Langer and Wellmann, 1990). Similarly, the light-dependent repair of CPDs in Arabidopsis also requires exposure to visible light prior to as well as after UV irradiation (Chen et al., 1994). Thus, the repair capacity of the plant depends on the quality and timing, as well as the quantity, of light in its environment. The influence of the environment on the steady-state level of pyrimidine dimers, the rate of induction of dimers, and the rate of photoreactivation of dimers has been illustrated in recent work on alfalfa (Takayanagi et al., 1994). These researchers found that seedlings grown in an essentially UV-free environment had the same steady-state levels of cyclobutyl dimers (approximately 6 dimers/megabase) as seedlings grown under unfiltered sunlight. In addition, a given dose of UV was found to induce significantly more dimers in the seedlings grown under artificial lighting, and these seedlings also had a lower rate of photoreactivation of CPDs than the identical strain grown under natural light. Thus, both the UV transparency and the repair capacity of higher plants is altered substantially in response to the ambient levels of UV and visible radiation. Similar effects have been observed in experiments that directly measure the effects of enhanced UV-B on yield (Caldwell et al., 1994).

In contrast to microbes and mammals, experimental evidence suggests that Arabidopsis may have a light-dependent pathway for the repair of pyrimidine (6–4) pyrimidinone photoproducts (Chen et al., 1994). Unlike the CPD-specific photolyase, this repair pathway does not require induction by prior exposure to visible light. It also does not require the *UVR1* gene product, which is essential for dark repair of 6–4 photoproducts (see below). Thus, Arabidopsis (and probably other plants) has the ability to photoreactivate both of the major UV-induced DNA damage products. Although photoreactivation of 6–4 photoproducts has not been observed in microbial or most animal systems, a 6–4 photoproduct-specific photolyase activity has been partially characterized in extracts of *Drosophila* larvae (Kim et al., 1994). If 6–4 photolyase activity exists in organisms as distantly related as plants and insects, it is important to determine whether it is expressed in other organisms. The discovery of the 6–4 photolyase is

particularly significant in that the biological effects of photoreactivation have previously been ascribed to the alleviation of the toxic effects of cyclobutyl dimers alone. A number of previously accepted conclusions about the role of CPDs in UV-induced toxicity may have to be reconsidered if we find that the assumptions underlying these conclusions are incorrect.

DARK REPAIR PATHWAYS

In contrast to photoreactivation, dark repair pathways do not directly reverse DNA damage, but instead replace the damaged DNA with new, undamaged nucleotides. These "excision repair" pathways fall into two major categories. Base excision repair involves the removal of a single damaged base through the action of one of many lesion-specific glycosylases, leaving the DNA sugar-phosphate backbone intact. The resulting abasic sites are then recognized by an apurinic/apyrimidinic endonuclease, which nicks the backbone of the DNA at the apurinic/apyrimidinic site (Sakumi and Sekiguchi, 1990). The nicked DNA is then restored to its original sequence through the combined actions of exonucleases, a repair polymerase, and DNA ligase. Nucleotide excision repair, exemplified by the UvrABC endonuclease of *E. coli*, differs from base excision repair in two ways; the spectrum of DNA damage products recognized by the UvrAB recognition complex is remarkably wide (Van Houten and Snowden, 1993), and the UvrBC endonuclease complex initiates removal of the damage by generating nicks at a specific distance 5′ and 3′ of the damaged base, which is then excised as an oligonucleotide through the action of the UvrD helicase (Fig. 3). It is interesting that excision repair of DNA damage has been shown to be coupled to transcription in both microbes and mammals (Hanawalt, 1994). Although repair occurs throughout the genome, repair of the transcribed strand of active genes occurs much more rapidly than repair overall. Many of the mammalian genes originally identified as DNA repair genes have recently been shown to encode proteins that have also been identified biochemically as transcription factors. Thus, strand-specific repair might be regarded as an additional UV-tolerance mechanism, because it enables the cell to resume transcribing DNA before any significant drop in the overall level of damage has occurred.

Repair pathways can remove and replace (A) or reverse (B) DNA damage

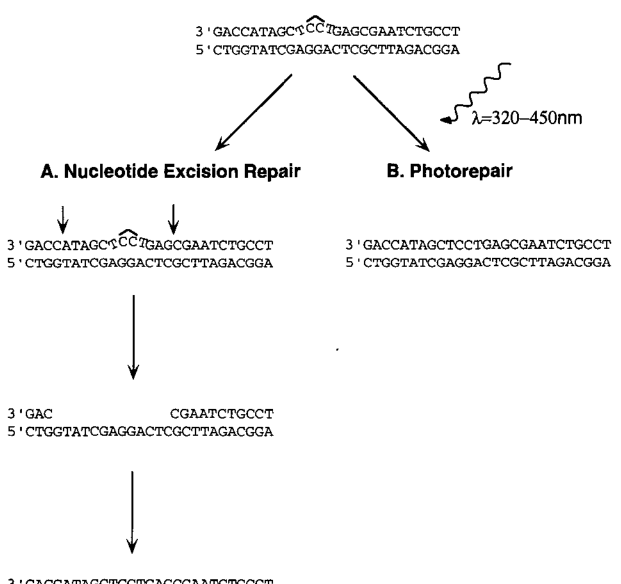

Figure 3. DNA repair pathways.

Excision repair of CPDs has been observed in several plant species. Early studies, reviewed by McLennan (1987), involved the use of a germicidal lamp to irradiate cell suspension cultures or protoplasts (for uniformity of UV penetration) producing high concentrations of CPDs. The disappearance of dimers from the nuclear fraction and their reappearance in the cytosol was measured by hydrolyzing the DNA and assaying, via TLC, the fraction of total thymidine bases that were present as dimers. The rate of dark repair of CPDs was found to vary widely from one plant system to another, with high rates of repair demonstrated for carrot suspension cultures (Howland, 1975) and protoplasts of carrot, *Haplopapus*, petunia, and tobacco (Howland and Hart, 1977), whereas excision repair of CPDs was undetectable in cultured soybean cells (Reilly and Klarman, 1980).

Recently, more sensitive techniques have been developed for the detection of UV-induced damage, including the use of lesion-specific antibodies (Mitchell and Rosenstein, 1987) and an exquisitely sensitive gel-electrophoresis-based method involving the extraction of intact DNA from plant cells, followed by cleavage of the DNA at CPDs, and finally the quantitative assay of various size classes of single-stranded DNAs to arrive at an average frequency of dimers (Quaite et al., 1994). These technical advances have enabled investigators to use relatively low doses of UV to study repair in intact plants. Dark repair rates for CPDs were recently assayed in Arabidopsis seedlings, where no significant repair of CPDs was detectable in 24 h, although repair of 6–4 photoproducts was efficient (Britt et al., 1993). In contrast, rapid dark repair of CPDs was observed in alfalfa (Quaite et al., 1994). Although Arabidopsis and alfalfa may actually differ in their capacity for dark repair, this disparity might also result from the differing experimental conditions employed by the two investigating groups. It has recently been demonstrated that excision repair in the alfalfa seedling, although efficient and easily detectable at high levels of initial UV damage, is undetectable at lower initial damage levels (Quaite et al., 1994). Extremely high doses of UV can also inhibit repair in plant tissues (Howland, 1975). Thus, although laboratory studies are essential for the determination of the biochemical basis of repair, caution must be used in extrapolating these results to make predictions concerning UV resistance in the field, where growth conditions, the plant tissues employed, and the levels of DNA damage induced by sunlight can radically affect both the extent of damage and the rate of repair.

It remains to be determined whether excision repair in plants occurs through the action of lesion-specific glycosylases or via a general excision repair pathway (or both). Several groups have published papers describing endonucleolytic activities obtained from plant extracts that exhibit some specificity for UV-irradiated DNA (Velemínsky et al., 1980; Doetsch et al., 1989; Murphy et al., 1993). Some of these activities are particularly intriguing in that they do not appear to recognize CPDs, suggesting that the recognition site may be the 6–4 photoproduct. In only one case (the endonuclease SP purified from spinach) has a plant UV-specific endonuclease been substantially purified and characterized; this enzyme was suggested to be a single-stranded endonuclease, which apparently recognizes a single-stranded region that is induced by 6–4 photoproducts but not by CPDs (Strickland et al., 1991).

CLASSICAL AND MOLECULAR GENETIC APPROACHES TO DNA REPAIR IN HIGHER PLANTS

A combination of classical, molecular, and biochemical approaches has been employed with great success to the study of the mechanisms and regulation of DNA repair and mutagenesis in *E. coli*. The genetics of DNA repair in yeast and mammals, although still incomplete, has recently provided valuable insights into various human repair-related diseases, as well as into the process of carcinogenesis (Cleaver, 1990, 1994). Although progress has been made in the genetics of UV repair in *Chlamydomonas* (Small, 1987), the study of the genetics of DNA repair in higher plants is still in its infancy. Using Arabidopsis as a model organism, as much for its minute (and therefore UV-B penetrable) seedlings as for its compact genome, several laboratories are currently attempting to identify the genes required for UV tolerance and repair. Arabidopsis cDNAs that may correspond to genes involved in repair were cloned by screening cDNA expression libraries for the ability to partially complement *E. coli* mutants defective in DNA repair (Pang et al., 1993; Santerre and Britt, 1994). Other groups have cloned Arabidopsis genes by screening for homology to previously isolated repair genes. This approach has yielded Arabidopsis homologs of photolyase (Batschauer, 1993) and topoisomerase I (Kieber et al., 1992), as well as a clone that includes a region of significant similarity to the *E. coli recA* gene (Cerutti et al., 1992). Several groups are currently screening for UV-sensitive mutants of Arabidopsis as a method of isolating mutants defective in DNA repair. This approach produced at least one mutant defective in the excision repair of 6–4 photoproducts (Britt et al., 1993), and other UV-sensitive mutants await biochemical characterization (Harlow et al., 1994). Because mutations in Arabidopsis can be cloned on the basis of their map position, and because cloned genes can be used to generate antisense "mutants" defective in repair, both the forward (repair-defective mutant to cloned gene) and reverse (cloned gene to repair defective antisense line) genetic approaches will be useful in determining the regulation, mechanism, and relative importance of each repair pathway in plant resistance to UV.

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