12.1.2?Photoinhibition and photoprotection

In light-response curves for photosynthesis (Figure 12.7), photosynthesis is light limited, as implied by the initial linear region of the curve. However, once a light-saturation point has been reached, further light exceeds the energy-utilising capacity of that photosynthesising leaf. Dashed lines in Figure 12.7 represent a continuation of the initial rate of light processing (quantum yield of photosynthesis) and show actual light absorption. The extent to which this absorbed light is not ‘gainfully employed’ for photosynthesis is set by $P_{\text{max}}$ (light-saturated rate of photosynthesis in normal air). At low light (< 100µmol quanta m$^{-2}$ s$^{-1}$), both sun and shade leaves use more than 80% of absorbed light for photosynthesis. Once $P_{\text{max}}$ has been reached, all additional light is in excess, and since shade plants have a lower $P_{\text{max}}$ than sun plants, they experience more excess light at a given photon irradiance above saturation. Additional stresses such as drought, nutrient limitation or temperature extremes can lead to a reduction in $P_{\text{max}}$ and thus increase the probability that plants will be exposed to excess light. However, even the most hardy sun plant will reach $P_{\text{max}}$ at less than full sunlight (incident beam normal to leaf surface). At that level (say, 1000µmol quanta m$^{-2}$ s$^{-1}$) approximately 25% of absorbed energy is used in driving photosynthesis, but at full sunlight (c. 2000µmol quanta m$^{-2}$ s$^{-1}$) as little as 10% is used in this way (Long et al. 1994). Individual leaves on plants growing in full sun commonly experience excess light. Such light is potentially damaging, and plants adapted to full sunlight have evolved with a number of mechanisms for either avoiding excess light or for dissipating excess absorbed energy.

Mechanisms for avoiding high light such as leaf angle and surface features, forestall absorption of excess light. Rapid responses, such as changes in leaf angle in *Oxalis* and *Omalanthus*, occur in a matter of minutes and can regulate light interception on a diurnal basis. Slower-acting mechanisms including production of wax on leaves will be useful where there has been a sustained change in light environment. These kinds of mechanisms constitute *external* photoprotection (Figure 12.8).
Figure 12.8 As photon irradiance is increased, energy utilisation gives way progressively to energy dissipation. Photosynthetic events shift from photochemistry, to external and then internal photoprotection and finally photodamage. These processes are interconnected and show considerable overlap. Their comparative importance for shade leaves and sun leaves is indicated. Photoprotection is especially well expressed in sun leaves acclimated to additional environmental and biotic stresses.

(Based on Demmig-Adams and Adams 1992; reproduced with permission of the Annual Review of Plant Physiology and Plant Molecular Biology)
Absorption of excess sunlight often leads to photoinhibition, defined here as a light-dependent downregulation of the quantum yield of photosynthesis (Figure 12.9). Ironic as it might seem, photoinhibition is one of the most important regulatory mechanisms in photosynthesis, and results from a series of internal photoprotective mechanisms which act to reduce the amount of light reaching the reaction centres of PSII. One immediate consequence is a reduction in quantum yield (expressed in terms of absorbed light).

Photoprotection (Figure 12.8) is normally sufficient to cope with light absorbed by leaves; photodamage only occurs when the capacity for photoprotection is exhausted. Photodamage is manifest as a decline in both quantum yield and $P_{\text{max}}$, and recovers only very slowly (hours to days), whereas photoinhibition recovers much faster (minutes to hours). Severe photodamage results in bleaching of pigments and damage to membranes (photo-oxidation) and may lead to tissue death.

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Consider the alternative fates of light energy absorbed by a leaf and their relevance to photoprotection, photoinhibition and photodamage (Figure 12.10). The proportion of absorbed energy consumed by these different sequences dictates their comparative significance, and in order of importance as protective devices they are:

1. assimilatory photochemistry, leading to fixation of CO\(_2\) into stable chemical products;

2. non-assimilatory photochemistry, that is, energy consumption by metabolic processes that do not result in fixation of CO\(_2\) into stable chemical products such as photorespiration, nitrate reduction and the Mehler reaction. All consume energy but there is no net gain in carbon as a result;

3. conversion of light energy into heat (thermal dissipation);

4. re-emission of photons as fluorescence. Emission of \textit{in vivo} Chl \textit{a} fluorescence is revealed dramatically during measurements (recall Figures 1.12, 1.13). Such emission still accounts for only c. 1% of energy derived from absorbed light.

Most of the NADPH and ATP formed during photosynthetic energy transduction is stored as stable photosynthetic products. Some is consumed in photorespiration and nitrate reduction. Because these non-assimilatory processes also utilise NADPH and ATP they help reduce the need for photoprotection. The Mehler reaction, in which electrons flow to O\(_2\) via photosynthetic system I (PSI) (Figure 1.10), still supports electron flow and thus might also reduce a need for photoprotection (Osmond and Grace 1995).
However, if photochemical capacity is exceeded by incoming energy, a plant will engage photoprotective mechanisms which increase the amount of energy dissipated as heat. This non-photochemical conversion of light energy is thought to occur in the PSII antennae and involves a group of pigments known as xanthophylls and including violaxanthin, antheraxanthin and zeaxanthin (Figure 12.11). These are a special group of carotenoids which undergo interconversion in response to excess light. Energy is dissipated in the process. In low light, violaxanthin predominates, but when light is in excess, conversion to zeaxanthin via antheraxanthin occurs (Figure 12.11). This conversion requires a low pH, ascorbate and NADPH. Such conditions exist in the lumen of chloroplasts in high light. When light levels are no longer excessive zeaxanthin slowly converts back to violaxanthin via antheraxanthin. Total pool sizes of the xanthophyll pigments increase with increasing exposure to excess light. Sun plants can have three- to four-fold larger pools of violaxanthin, antheraxanthin and zeaxanthin than shade plants and the presence of other stresses can also result in increases in pool size.

Internal differences between sun and shade leaves with respect to energy dissipation are also apparent in different patterns of attenuation of light through mesophyll tissues. This is nicely illustrated by *Cotyledon orbiculata*, the CAM plant cited earlier (Figure 12.5). Here, xanthophylls are mostly found in outermost cell layers where the light environment is strongest. If the reflective wax coating is intact no internal photoprotection is required at growth irradiance and there is no zeaxanthin formed. However, if surface wax (external photo-protection) is removed by hand, internal photoprotection is then needed and zeaxanthin appears in the outermost layer (Figure 12.12).
The presence of antheraxanthin and zeaxanthin has been well correlated with increasing thermal dissipation of absorbed light energy. If conversion of violaxanthin to antheraxanthin and zeaxanthin is prevented by treating leaves with DTT (dithiothreitol), then development of thermal dissipation is reduced and the probability of photodamage is increased. The exact mechanism by which zeaxanthin mediates an increase in thermal dissipation is not clear, but it is definitely linked to a pH gradient across the thylakoid membranes (pH from H⁺ pumping in Figure 1.11). This link to pH makes the mechanism very sensitive to changes in light because pH can be dissipated very quickly in low light, whereas reconversion of zeaxanthin to violaxanthin is relatively slow (discussed below in connection with sunflecks).

Traditionally, photodamage has been associated with damage to the D1 protein of PSII (Section 1.2). Most of that research comes from unicellular green algae where photodamage occurs if D1 degradation is faster than D1 synthesis. However, in higher plants, D1 protein turnover is optimal at growth irradiance and declines at both lower and higher irradiances. When D1 damage occurs in higher plants PSII centres become photochemically inactive. Non-functional PSII centres may then accumulate in the chloroplast granum and somehow increase thermal dissipation of excess light energy (Anderson and Aro 1994). Shade plants with their larger granal stacks may have greater capacity to accumulate non-functional PSII centres, whereas sun plants may rely on a higher rate of D1 turnover and larger xanthophyll pool sizes for their internal photoprotection.

In either circumstance, photoprotection involves a prodigious turnover of PSII reaction centres. W.S. Chow (pers. comm.) estimates that about 1 million damaged centres are renewed every second for each square millimetre of a capsicum leaf, even in low light (20µmol quanta m⁻² s⁻¹, i.e. near to light-compensation point). At light saturation (say, 1000µmol quanta m⁻² s⁻¹), turnover would be about 50 million reaction centres per second! Self-repair on this scale is a prerequisite to continuing leaf function, and a constant drain on plant resources because replacement of D1 protein is part of the repair process.
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